

LIPID PROFILE IN MALNUTRITION

**THESIS
FOR
DOCTOR OF MEDICINE
(PAEDIATRICS)**



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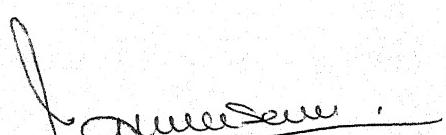
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C E R T I F I C A T E

This is to certify that the work in connection
with thesis of DR. ASHOK KUMAR on "LIPID PROFILE IN
MALNUTRITION" for M.D. (Paediatrics) of Bundalkhand
University was conducted in the department of Paediatrics.

He has put in the necessary stay in the
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Dated : 27.5.1983

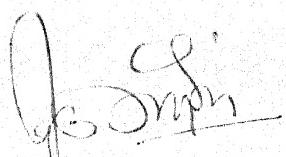

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C E R T I F I C A T E

This is to certify that the work on " LIPID PROFILE IN MALNUTRITION " which is being submitted for M.D. (Paediatrics) thesis by DR. ASHOK KUMAR has been carried out under my guidance and supervision in the department of Paediatrics. The techniques embodied in the thesis were undertaken by the candidate himself and the observations recorded have been periodically checked by me.

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C E R T I F I C A T E

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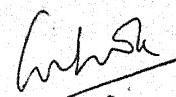
I am highly grateful to Dr. R.S.Sethi, M.D., D.C.H., Lecturer in the department of Paediatrics, M.L.B. Medical College, Jhansi who was one of my most sincere helpers fulfilling all my needs and curiosities.

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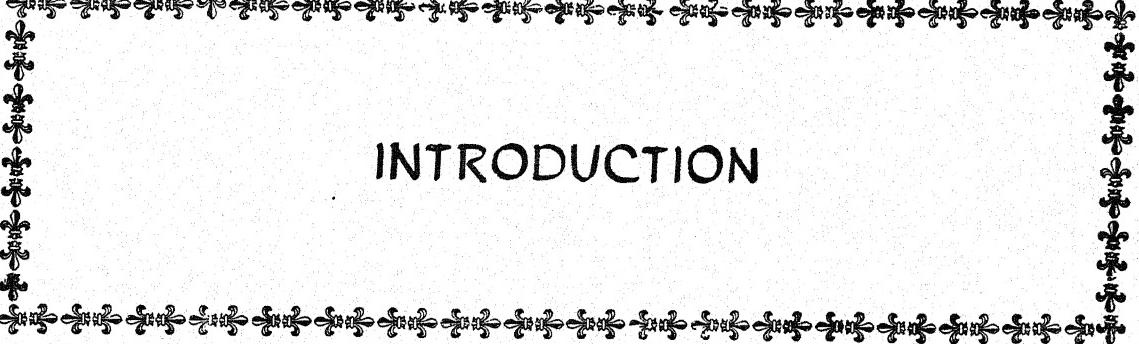
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INTRODUCTION

INTRODUCTION

Never before in the history, the human race has faced a situation as it does today. It now stands at a point where millions in the developing countries suffer from perpetual hunger. Many die every day directly or indirectly from malnutrition. Children are the worst victims; and kwashiorkor and marasmus are the pitiable symbols of this immense human misery.

In India, according to the latest 1981 census preschool children upto 5 years constitute about 116 million population. About 19 million children are added to the pool every year. Out of 23 million children born every year, 4 million die in childhood, 3 million become healthy and productive citizen but rest 16 million become adults with poor mental and physical abilities due to serious undernutrition in their childhood.

The term protein caloric malnutrition comprises a variety of very closely interrelated clinical syndromes, which are the result of differences in the severity and duration of the deficiencies, and the age

of the child and the relative importance of the protein deficiency in regard to that of calories.

In our country, the incidence of severe form of Protein Calorie Malnutrition like kwashiorkor and marasmus is about 1-2 %, whereas the mild and moderate forms account for 60-80 % of total preschool population.

It is generally accepted that malnutrition is the result of protein and / or calorie deficiency . There is no doubt that the diets of those children in whom malnutrition develops are also frequently low in fat content. Further the well documented observation that the fatty infiltration of the liver and good layer of subcutaneous fat are characteristic signs of one of the severe form of malnutrition i.e. kwashiorkor, indicate a defect in fat metabolism.

There have been an increasing volume of investigations into the metabolic dysfunction occurring in protein calorie malnutrition in infants, young children and in experimental animals. In particular research into protein metabolism, carbohydrate metabolism, enzyme activities, body composition (including water and electrolytes) and lipid metabolism have not only increased the fundamental knowledge , but has already been of practical value

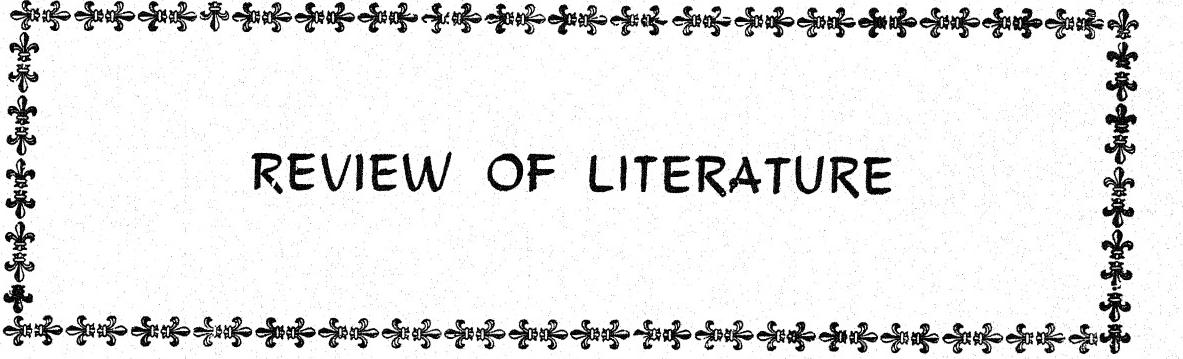
in indicating improved methods of treatment. Also, the researches have suggested possible approaches for measuring the severity or degree of protein malnutrition quantitatively and specially of diagnosing less severe degrees of deficiency biochemically.

Some workers have reported differences in kwashiorkor and marasmus group in total lipid levels and lipid fractions. In untreated kwashiorkor the total lipids and other fractions are low with increased free fatty acid levels in serum, whereas in marasmus, the serum lipids remain within normal limits.

The majority of lipid investigations have been carried out in kwashiorkor group, while studies on lipid metabolism in marasmus and other malnutrition groups are very few. In the present study, though a humble one , the characteristics of serum Total lipids and its fractions(Total cholesterol and free fatty acids) in the severe and recovery phases of undernutrition, marasmus, marasmic kwashiorkor and kwashierker have been investigated.

It was aimed to evaluate the lipid profile in infants and children with varying degrees of malnutrition to assess the status of total lipids and

its fractions (total cholesterol and free fatty acids) in relation to severity of malnutrition and to further evaluate the possible interrelationship between the clinical progress of the malnourished child and subsequent changes in the lipid profile by serially following the cases.



REVIEW OF LITERATURE

REVIEW OF LITERATURE

The resultant of interaction between man and his environment is health or disease. Certain ecological, biological and socio-economical conditions of the developing countries of the world favour the greater frequency and severity of certain pathological conditions, foremost among them being the nutritional deficiencies. Infantile malnutrition due to protein and calorie deficiency must have been common in most of the civilizations of the world for centuries, but attention has only been focussed upon it in the early years of this century. Historically marasmus (Greek - Marasmus = wasting) was recognised for ages as being a major contributor to high infant mortality. Proctor (1926-27) and other workers described the physical appearances of the malnourished children but nutrition was not thought to be the cause of such appearance. Williams (1931-32-33) made a great contribution to the subject when she gave a name ' Kwashiorkor ', derived from Ga language of Ghana, to a tropical syndrome and maintained that it was nutritional in origin.

and that it was different from pellagra. This was the disease first child got when the second was on the way. It was characterized by skin and hair changes, oedema, moon face, fatty liver, hypoalbuminaemia and psychomotor changes. Waterlow (1948) and Jelliffe et al (1954) used the term ' Sugar baby ' to describe , obviously similar condition to Kwashiorkor , found in West Indies, where dermatosis was uncommon though oedema was prominent.

After the second world war (1939-45) the workers were conversant with the fact that marasmus and kwashiorkor were two syndromes of nutritional deficiency. Waterlow (1948) in his monograph on 'fatty liver diseases in infants' recognised that " Kwashiorkor was a nutritional disease probably due to a deficiency of protein and that, in babies dying of under nutrition things were very different, there was complete loss of subcutaneous fat and only small amounts in the liver. " He further added that ' the two syndromes might not be rigidly distinct but that one could be converted into the other by increasing or decreasing the caloric intake.'

Jelliffe (1959) coined the term ' Protein Calorie Malnutrition (PCM) of early childhood ' to

include the mild, moderate and severe degrees of malnutrition, which was later on accepted by FAO/WHO Joint Expert Committee on Nutrition (1962) to cover diseases such as marasmus, kwashiorkor and famine oedema. There was a short lived effort through WHO to introduce the term ' Protein Calorie Deficiency Diseases ', but this was abandoned by the expert group meeting in 1970 in favour of PCM. Joule being the unit of energy measurement, there were lots of proposals to replace the term calorie by joule and this finally led to a general use of the term ' Protein Energy Malnutrition ' (PEM) rather than Protein Calorie Malnutrition.

To emphasize the energy crisis all over the world and the nutritional deficiency being a part of it, some workers have used the term ' Energy Protein Malnutrition ' (EPM) - McLaren (1973).

To combat malnutrition grading of PEM was necessary for defining the priorities and to formulate therapy in individual patients. Gomez, et al (1985) are credited with the first ever classification of malnutrition. They used the actual weight expressed as a percentage of standard weight (Boston, 50th percentile) for that age. Children with more than

90 % of expected weight for age were classified as normal, between 89 % and 75 % of expected weight were in mild grade , between 74 % and 60 % of expected weight were in moderate grade and children of less than 60 % of expected weight were classified as severely malnourished. In this classification , presence or absence of oedema was not taken into consideration. The main drawbacks of this classification were that it assumed all children of certain age to have the same weight irrespective of their size, and it also included such children who were underweight as a result of malnutrition in the past.

Jelliffe (1966) modified the Gomez classification and included all cases with nutritional oedema, irrespective of weight in severe degree.

Mc Laren (1967) introduced a scoring system for classifying the severe forms of malnutrition only (weight of children \leq 75 % of 50th percentile of Boston standard), based on all the three methods of assessment viz. clinical, anthropometric and biochemical. He showed that the severe degree of malnutrition in its various clinical forms of marasmus, marasmic kwashiorkor and kwashiorkor formed a spectrum of both clinical signs and biochemical changes, both

being most marked in full blown kwashiorkor and least evident in pure marasmus. In this classification also, the problem of expressing chronicity and stage of disease, however remained unsolved.

Arnold (1969) devised a ' Quacstick method ' based upon height and mid arm circumference. The advantages of this classification were ; it was age independent, good for field surveys and could be applied by an unskilled personnel. Based on this classification, children were divided into two broad categories ' Mal-nourished ' and ' Normal. '

Kanawati and Mc Laren (1970), proposed that the ratio of mid arm circumference and head circumference was independent of sex and age atleast from 3 to 48 months. If the ratio was ≥ 0.310 , children were grouped as nutritionally healthy. Children with ratio between 0.310 and 0.280 were classified in mild PCM group, children with ratio between 0.279 and 0.250 were put in moderate PCM group, and children with ratio ≤ 0.249 were classified in severe PCM group. This classification was again useful for screening large number and could not be used for an individual child.

In 1970, Rao et al suggested an age independent

classification by using weight in kg and height in cms.

Weight in kg

(Height in cms.)²

Values less than 0.6016 reflected early PEM.

The classification prepared by ' WellCome Trust' was approved by FAO/WHO expert committee in 1971. In this classification, children were grouped in 5 grades using their weight as percent of 50th percentile of Harvard standard value.

Under weight - Children having body weight between 80% and 60 % of 50th percentile of Harvard standard, without oedema and with minimal deficit in weight for height.

Nutritional dwarf - Children having body weight less than 60 % of 50th percentile of Harvard standard, without oedema and with minimal deficit in weight for height.

Marmasmus - Children weighing less than 60 % of 50th percentile of Harvard standard, without oedema and with ' + +' deficit in weight for height.

Kwashiorkor - Children weighing between 80 % and 60 % of 50th percentile of Harvard Standard, with oedema and with ' + +' deficit in weight for height.

Marasmic kwashiorkor - Children weighing less than 60% of the 50th percentile of Harvard standard, with oedema and with ' + + ' deficit in weight for height.

$$\text{* weight for height} = \frac{\text{Weight of patient}}{\text{Weight of normal subject of same height.}} \times 100$$

This was probably the first classification in which an attempt was made to use weight/height as well as weight/age ratios, and included a separate category of 'Nutritional dwarfs'. However, some drawbacks of this classification were ; it confused between the type and severity of malnutrition, in this system kwashiorkor appeared to be less severe than marasmus and marasmic kwashiorkor as body weight was between 60 % and 80 %, gradation of deficit in weight for height by such terms used as ' minimal' or ' + + ' could not be quantitated.

Nutrition Sub-committee of the Indian Academy of Paediatrics (1972) classified PCM into 4 grades using 50th percentile of Harvard growth standards as a reference point.

Grade I. Children having weight between 80 % and 71 % of reference standard.

Grade II. Children weighing between 70 % and 61 % of reference standard.

Grade III. Children weighing between 60 % and 51% of reference standard.

Grade IV. Children having weight less than 50 % of reference standard.

Grade I and II were underweight and grade III and IV corresponded to marasmus. When nutritional oedema was present, letter K was suffixed to the grade denoting malnutrition e.g. grade I K, II K , III K etc. I K and II K meant kwashiorkor and grade III K and IV K corresponded to marasmic kwashiorkor.

Waterlow and Rutishauser (1974), published a classification based on weight and height . The present malnutrition was called ' wasting ' , as measured by loss of weight in relation to height, and past malnutrition was called ' Stunting ' , seen as low weight for age ratios. The children were grouped in following grades :

Healthy children - Weight for height more than 90 % and height for age more than 95 %.

Grade 1. Weight for height between 90 % and 80 % and height for age between 95 % and 90 %.

Grade 2. Weight for height between 80 % and 70 % and height for age between 89 % and 85 %.

Grade 3. Weight for height less than 70 % and height for age less than 85 %.

Waterlow and Rutishauser found that weight for height was independent of age in the age group of 1 to 4 years.

LIPIDS

The term lipid is used to describe collectively cholesterol, glycerides (neutral fats), phospholipids, glycolipids, free fatty acids and fat soluble vitamins circulating in the blood. The lipids circulate in the blood in combination with certain proteins as macromolecules, known as lipo proteins. (Hawks, 1976).

Cholesterol is a sterol containing hydrogenated phanthrene ring. 70 to 80 percent of serum total cholesterol is in ester form and 20 to 30 % form free cholesterol. (Schoenheimer and Sperry, 1934). Gleuk and Tsang (1972), determined the concentration of cholesterol in cord blood samples and found mean level to be 63.8 ± 18.7 mg / dl. Owen and Lubin (1971), found out the mean cholesterol level in the age group

of 1 to 6 years of age, as 159 mg/dl, for 1 to 2 years old children and 165 mg/dl for 2 to 6 years of age.

Glycerides in particular Triglycerides forms the main bulk of dietary lipid. About 1 to 2 gm/kg of body weight of glycerides are ingested daily (Henry, 1977). These are the esters of long chain fatty acids and glycerol. During metabolism they are broken into di-and monoglycerides and fatty acids. After absorption, the fatty acids are again converted into triglycerides which are deposited in the liver and adipose tissue (Rex Montgomery, 1977).

Free fatty acids forms about 5 % of total serum fatty acids. This is the form in which fat enters the blood from the storage depots in adipose tissue to serve as fuel for the tissues (Rex Montgomery, 1977). The concentration of free fatty acids in the plasma is very sensitive to nutritional state. After a meal or a test dose of glucose or an injection of insulin, the level is reduced to half or less (Hadden , 1967) and during starvation free fatty acids level rises (Lewis et al, 1964 , 1966) .

Nigam et al, 1983 and Sharma et al, 1983, determined the FFA levels in cord blood samples and found the mean level to be between 0.30 and 0.45 mEq/l. The normal level of free fatty acids in serum is 400 to 900 μ eq/l (Millian Novak , 1965).

Lipo proteins is the form in which most of the lipids are present in plasma. Nearly all the cholesterol, phospholipid and endogenous triglycerides in blood are present as aggregates with various protein moieties (Hawks, 1976). Following are the major lipo proteins groups -

Chylomicrons.

very Low Density Lipo proteins (VLDL)

Low Density Lipo proteins (LDL)

High Density Lipo proteins (HDL)

Hawks, (1976) described phospholipids as a group of compounds, which are composed of glycerol, fatty acids and phosphoric acid. These are found in blood cells, plasma and all tissue cells, usually in combination with proteins and other lipids.

MALNUTRITION AND TOTAL LIPIDS

van Der Sar (1951) studied 16 hospitalized kwashiorkor children and estimated serum total lipids in them before and after putting them on dietary therapy. He found variable values of serum total lipids on the day of admission. In 6 children the values were between 360 and 635 mg/dl, in 5 children the values were between 667 and 855 mg/dl and in 5 cases, the values were between 930 and 1490 mg/dl. The repeat estimations at the time of discharge from hospital also showed variable results. 4 children showed a rise and 6 children showed a fall in serum total lipid levels.

Schwartz and Dean (1957) investigated 20 hospitalized kwashiorkor children between 15 and 30 months to investigate serum total lipid levels on the day of admission and then after putting them on nutrition therapy, every week. They found a significant rise in the levels, reaching upto 969 mg/dl after 10 days of dietary therapy, as compared to the admission level 532 mg/dl. After 6 weeks time the level fell to 644 mg/dl, which was still higher than the initial level. They divided children into 2 groups,

in one group fat was supplemented in diet, and in other no fat supplementation was done. They found no significant change in the levels. Schwartz and Dean proposed that probably the endogenous fat mobilization led to high levels after the diet therapy.

Cravioto, et al (1959), noted similar changes in the levels of serum total lipids in kwashiorkor cases. They correlated the changes occurring in malnourished children with those of normal children and found a great similarity. They suggested that change in kwashiorkor cases was a biochemical expression of the normal process of growth and development, which arrested during the course of malnutrition, reassumed a rate and pattern comparable to those of normal children .

Mac Donald et al (1963) , studied kwashiorkor children between the ages of 13 months and 3 years for the levels of total lipids and its fractions at the time of admission, on 10th day and on 20th day of diet therapy. They found a significant rise in serum total lipid levels after 10 days of treatment followed by a fall in next 10 days period and this rise in the total lipids level was due to an increase in cholesterol, glycerides and phospholipids.

They suggested 2 important features to distinguish kwashiorkor from marasmus ; one was very fatty liver , and the other was normal or slightly depleted quantities of depot fat. They proposed that the lipid in those sites most probably arose from the dietary carbohydrate and the changes in lipid levels during treatment reflected a reversal of changes leading to their formation.

Gurson et al (1973), studied serum total lipids and its fractions in marasmus cases with a mean age of 10.5 months and mean weight of 4.50 kg. They estimated serum total lipids on the day of admission and on 30th day after dietary treatment. They noted no significant change in lipid levels as 0 day levels were 647 ± 67 mg/dl and 30 days levels were 645 ± 74 mg/dl, as compared to control levels of 607 ± 74 mg/dl.

MALNUTRITION AND SERUM TOTAL CHOLESTEROL

The workers from Nutrition Research laboratory, Coonoor, India (1952) studied hepatic cholesterol content in kwashiorkor cases, and found a moderate increase in cholesterol content in liver at the time of admission, which regressed after diet therapy.

Van Der Sar (1951) noted significantly low levels of serum total cholesterol on admission in kwashiorkor, which increased after two to three weeks of diet therapy. The found that rise in serum total cholesterol level was principally due to increased esterified fraction.

Dean and Schwartz (1953) studied total and esterified cholesterol level in kwashiorkor cases and found a decreased cholesterol levels on admission with a tendency to rise and fall after dietary therapy. They could not attribute the changes in cholesterol levels to quality or quantity of diet ingested. Therefore, they suggested that the rapid rise in levels was due to release of preformed fat and cholesterol from stores.

Workers of Nutrition Research laboratory, Coenra (1954), found considerably low levels of total cholesterol in kwashiorkor cases which increased significantly after 3 weeks of dietary therapy. They found the average ratio of free to total cholesterol as 0.40 at the time of admission as compared to normal ratio of 0.30.

Ramnathan (1955), studied convalescing kwashiorkor cases and made similar observations as

found by previous workers (van Der Sar, 1951; Dean and Schwartz, 1953; Coonoor study, 1954).

Schwartz and Dean (1957), in their further study found that the total cholesterol level came down to initial level after about 3 weeks therapy, whereas the esterified cholesterol level remained high.

Cravioto et al (1959), also noted low levels of total cholesterol in kwashiorker cases, at admission time which rose significantly to a maximum level and then either levelled off or decreased after starting the dietary therapy. They compared the changes with those occurring in normal children from birth to the end of childhood and found a striking resemblance. They suggested that the changes noted during the initial recovery of a malnourished child whose rate of growth and development were decreased to a point at which clinically and biochemically he no longer resembled a child of his own chronologic age but practically a new born, were similar to changes noted in normal new borns as part of their process of maturation inherent to normal growth and development.

Schandl and Hensen (1961), Mac Donald et al (1963), made similar observations as noted by

previous workers (van Der Sar, 1951 ; Dean and Schwartz, 1953 ; Coonoor study, 1954 ; Ramnathan , 1955

Schendel Hansen emphasized that the arrested rise or fall was associated with either the onset of complication or inadequate therapy.

Lewis et al (1964), noted a significant rise in levels in total cholesterol in kwashiorkor cases from 68 ± 21 mg/dl to 206 ± 113 mg/dl after 2 weeks of dietary therapy, which fell to 152 ± 29 mg/dl after 3 weeks of treatment , the later value was still significantly higher than the initial value. In marasmus cases, they found no significant change in total cholesterol levels even after 3 weeks of dietary therapy. The level after therapy was 143 ± 50 mg/dl as compared to the admission level 139 ± 49 mg/dl.

Jaya Rao and Krishna Prasad (1966), made similar observations in 85 kwashiorkor cases as noted by Lewis et al(1964).

Taylor (1971), also made similar observations of significant rise in total cholesterol levels after dietary therapy in kwashiorkor cases. In contrast to previous workers he did not notice any significant fall at 3 weeks.

Debnath (1972) , studied kwashiorkor and marasmus cases alongwith complicated kwashiorkor and marasmus cases in the age group of 8 to 60 months to evaluate serum total cholesterol levels on the day of admission, on 10th day and on 30th day of dietary therapy. In uncomplicated kwashiorkor cases, he noted a significant rise in the levels, which rose from 145.9 mg/dl to 189.9 mg/dl on 10th day but on 30th day the level fell back to 155.6 mg/dl which was slightly higher than the initial level. In marasmus cases, in contrast to kwashiorker he found a less pronounced gradual rise in mean cholesterol level from 135.3 mg /dl at the time of admission to 155.7 mg/dl on 10th day and 169.8 mg/dl on 30th day of dietary treatment. In the complicated group (malnutrition with other diseases) he did not observe any significant change in levels from the time of admission to 30 days of therapy.

Debnath suggested that in face of fatty liver and good depot fat, the low amount of cholesterol in untreated cases of kwashiorkor could be due to lack of appropriate proteins acceptors necessary for discharging lipid from the liver in the form of lipoproteins.

Thus lack of protein acceptors together with the lipogenic tendency, could account for the development of a fatty liver that was responsive to the addition of proteins to the diet.

Gurson, et al (1973), studied marasmic children with mean age of 10.5 months and mean weight of 4.50 kg in the severe and recovery phases to evaluate various lipid fractions. They found the mean level of total cholesterol at admission was 180 mg/dl as compared to level of 172 mg/dl in control cases. After 30 days of therapy, they found the level was 164 mg/dl which was not significantly different from the initial level or the control level. They concluded that similar to mechanisms controlling protein and carbohydrate homeostasis, the marasmic infants probably maintain a balanced source for lipids, as a result of which the plasma levels of lipid and lipid fractions remain within normal limit.

MALNUTRITION AND SERUM FREE FATTY ACIDS

Lewis, et al (1964), studied the levels of free fatty acids in kwashiorkor and marasmus cases and compared the values with normal subjects. The mean age

for kwashiorkor, marasmus and control groups were 22 months, 18 months and 23 months respectively. They found a significantly raised free fatty acid (FFA) values in kwashiorkor cases to $914 \pm 372 \mu\text{eq/l}$, as compared to control values of $367 \pm 306 \mu\text{eq/l}$ ($P < 0.001$) at the time of admission. During treatment rapid fall in FFA level was observed by them and at the end of about 3 weeks time, the values were comparable to those of control group i.e. $402 \pm 336 \mu\text{eq/l}$.

In two of the patients only carbohydrate diet was given for about seven days and the plasma FFA level fell significantly. After introducing proteins in diet in the cases, the levels tended to rise to some extent but eventually showed a fall after five to six days. They concluded that glucose administration depressed FFA levels, whereas prolonged starvation caused elevation in plasma levels. They found the mean level of plasma FFA on admission as $815 \pm 347 \mu\text{eq/l}$ in marasmus, which subsequently fell to $97 \pm 31 \mu\text{eq/l}$ after initiation of dietary treatment, and finally at the time of discharge, it again rose to $466 \pm 104 \mu\text{eq/l}$.

Jaya Rao and Krishna Prasad (1966) studied 85 kwashiorker children aged 18 to 48 months and weighing

between 6.3 and 14.8 kg. They found that after nutritional therapy with 6 gms proteins and 200 Calories per kg per day, there was a significant fall in mean value of non-esterified fatty acids (NEFA) from the pretreatment value of $875.5 \pm 47.16 \mu\text{eq/l}$ to $331.6 \pm 94.75 \mu\text{eq/l}$ on 30th day. The mean concentration of NEFA in their control group was $466 \pm 43.16 \mu\text{eq/l}$.

They also studied the effect of epinephrine on the levels of NEFA. At the time of admission, after epinephrine stimulation NEFA levels increased from $875.5 \pm 47.16 \mu\text{eq/l}$ to $1,129.9 \pm 94.52 \mu\text{eq/l}$ at 30 min and $1,357.2 \pm 273.62 \mu\text{eq/l}$ at 60 min time. Corresponding levels after therapy were 993.0 ± 164.51 and $493.0 \pm 126.4 \mu\text{eq/l}$. The mean maximal response to epinephrine administered on admission and after treatment were not statistically different from each other. They suggested that the high circulating NEFA levels could be due to hepatic damage in kwashiorkor cases causing defective uptake of NEFA, or that increased NEFA levels represented an alternative source of energy to tissues.

Plasma FFA concentrations were increased in moderate to severe cases of kwashiorkor (Lewis, et al 1964).

This observation was somewhat unexpected in view of the belief that infants developing kwashiorkor habitually consumed an excess of dietary carbohydrates, and it was a well established fact that plasma FFA levels were depressed by the intake of carbohydrates (Lewis , et al, 1964). Lewis , et al (1966), further studied FFA flux through plasma to assess the quantitative release of FFA into plasma in malnourished children. They studied 4 kwashiorkor and 2 marasmus cases. In the control subjects the flux rate was found to be in the range of 4.9 to 10.9 $\mu\text{eq}/\text{min}/\text{kg}$ body weight with the mean value of 6.6 $\mu\text{eq}/\text{min}/\text{kg}$, and in the kwashiorkor cases the flux rate was 22.6 to 107.5 $\mu\text{eq}/\text{min}/\text{kg}$ with its mean as 64.3 $\mu\text{eq}/\text{min}/\text{kg}$ on admission to the hospital. They found that the mean flux in kwashiorkor cases was about 10 times than that of control group. After treatment for about 10 days, the FFA flux in kwashiorkor had fallen steeply in all patients to 8.1 to 13.4 $\mu\text{eq}/\text{min}/\text{kg}$ with the mean value as 10.6 $\mu\text{eq}/\text{min}/\text{kg}$ body weight. In the marasmus group, the patients had flux rates above the normal range i.e. 26.6 $\mu\text{eq}/\text{min}/\text{kg}$.

In their study , Lewis and other workers found the mean FFA concentrations in kwashiorkor ,

marasmus and control cases were 1049.25 ± 296.420 , 899.5 ± 362.745 and $287.667 \pm 100.271 \mu\text{eq}/\text{l}$ respectively. Their study showed that, though the plasma FFA level was 3 to 4 times of the normal in kwashiorkor cases, but the mean FFA flux rate was as much as 10 times higher than the control group. They concluded that production and removal of FFA was increased in kwashiorkor and marasmus.

Hadden (1967), studied the inter relationship between blood glucose , FFA and insulin levels in 24 kwashiorkor cases and 9 marasmus cases, and investigated them on admission and then regularly at an interval of 4 days during the first two weeks of dietary therapy. He observed no significant change in FFA levels in kwashiorkor cases after 2 weeks therapy, as mean level at admission was $1.04 \pm 0.08 \text{ mEq/l}$ which fell to $0.75 \pm 0.06 \text{ mEq/l}$. On the other hand in marasmus, the mean FFA value fluctuated between 0.70 ± 0.16 and $0.91 \pm 0.10 \text{ mEq/l}$. He further observed that the kwashiorkor cases showed temporary impairment of carbohydrate tolerance, associated with elevated circulating FFA and a delayed fall in FFA, following a glucose load in the diets. He suggested that their

metabolism could be partially blocked, perhaps due to some enzyme deficiency at the entry to the Kreb-cycle , and stressed that if it were so, this could explain the accumulation of body fat both in depots and in the liver.

Gurson and Saner (1969), studied 14 marasmus cases on admission (within 11 days), after 6 weeks and finally after 14 weeks of dietary treatment. They found no significant change in FFA levels in marasmic cases. In their later study of FFA in marasmic cases (1973), they concluded that marasmic cases probably maintain a balanced source of lipids as a result of which, the plasma level of total lipid and lipid fractions except lipoproteins and phospholipids, remained within normal limit.

Similarly Agbedana, et al (1979), found no significant changes in FFA values in untreated marasmic cases, though the values were significantly higher in untreated kwashiorkor cases.

MALNUTRITION AND OTHER LIPID FRACTIONS

Schwartz and Dean (1957), found a significant rise in serum phospholipid levels from 145 mg % to 206 mg % dl at the end of 2 weeks of dietary therapy in kwashiorkor cases, which subsequently fell to 180 mg/dl after 4 weeks therapy. Mac Donald, et al (1963) and Lewis, et al (1964), made a similar observation in kwashiorkor children. However, in marasmus, Lewis, et al observed a rise in serum phospholipid levels after 2 weeks of dietary therapy, though it was not significant.

Taylor (1971), found a gradual rise in levels of phospholipids in kwashiorkor cases after 20 days of therapy after which, he observed a fall on 30th day, though the values were still higher than the initial level. Gursen, et al (1973), studied serum phospholipid levels in marasmic cases, and they found a significant fall in levels after dietary therapy from 150 ± 24 mg/dl to 117 ± 8.9 mg/dl.

Mac Donald, et al (1963), studied serum triglyceride levels at the time of admission and after dietary therapy in kwashiorkor cases and found a significant rise in levels on 10th day of therapy as levels rose from 90 ± 36 mg/dl to a level of 203 ± 46 mg/dl.

and again fell to the initial level of 93 ± 37 mg/dl on 20th day of therapy. Lewis , et al (1964) and Jaya Rao and Krishna Prasad (1966), found similar changes in kwashiorkor cases. Whereas in marasmus , Lewis,et al found no significant change on 10th day of therapy, as the levels were 160 ± 65 mg/dl as compared to 150 ± 57 mg/dl on admission but on 20th day of therapy, they noted a significant fall to a level of 85 ± 14 mg/dl. They compared the levels in marasmus and kwashiorkor cases and noted a higher level in marasmus, the cause they suggested , was large production of endogenous plasma triglycerides from circulating FFA, whereas in kwashiorkor cases the lipid accumulated in the liver as liver was unable to dispose of fatty acids adequately.

Truswell and Hansen (1969), and Taylor (1971),noted similar changes in triglyceride levels in kwashiorkor cases as noted by previous workers Mac Donald, 1963 ; Lewis et al, 1964 ; Jaya Rao and Krishna Prasad, 1966) , but Taylor found that the raised levels persisted upto 20th day of therapy and then fell to initial level. Agbedana (1979),studied

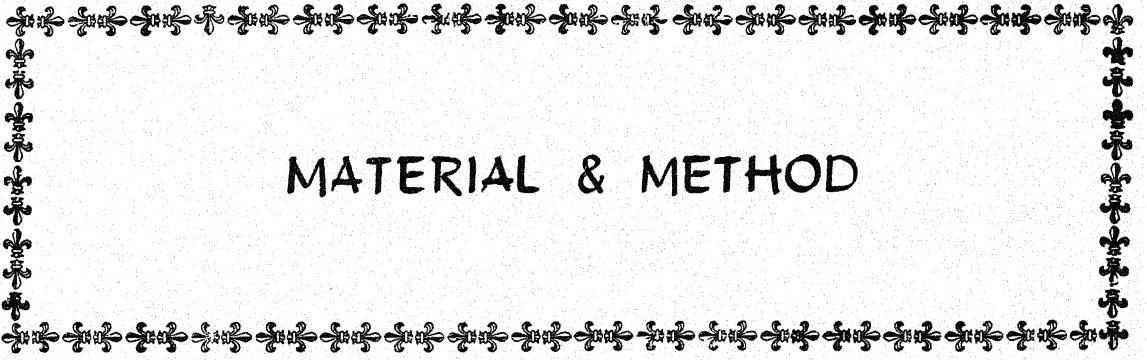
triglyceride levels in kwashiorkor and marasmus cases in the severe stage and found slightly raised levels of 114 ± 19 mg/dl in kwashiorkor as compared to control levels of 90 ± 6.0 mg/dl, but this difference was not significant. In marasmic cases the levels of 98 ± 19 mg/dl were similar to control cases.

Cravioto, et al (1959), studied serum lipoprotein levels in kwashiorkor cases and found significantly low levels of alpha and beta lipoproteins at admission time, which rose after dietary therapy reaching to a maximum and then, either levelled off or decreased. They also found that in two of the cases the initial levels of beta lipoproteins were either normal or higher than normal and after therapy the levels decreased. They further correlated these changes with the changes occurring in normal children from birth to the end of childhood, and noted a similarity. They concluded that the biochemical changes, independent of age, sex and severity of mal-nutrition were normal finding in much younger well nourished children.

Chatterjee and Chaudhuri (1961) also reported significantly low levels of alpha lipoproteins

in PCM cases at the severe stage of disease. Truswell , et al (1969) , reported variable levels of alpha lipoproteins in kwashiorkor cases at admission and commented that the electrophoresis technique, by which the previous workers had reported low levels, was not a reliable method. Gurson, et al (1973) , studied marasmic cases and found significantly low fractions of alpha lipoproteins as $24 \pm 8.3\%$ of total lipoproteins, as compared to the normal control fraction of $32 \pm 9\%$. They noted a rise in alpha lipoproteins fraction as recovery occurred . They did not find any change in the beta lipoprotein fractions either at the initial phase or after therapy as compared to the control fraction. They explained the low fraction of alpha lipoproteins in their study were due to significantly low levels of albumin in plasma as compared to the normal control levels.

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MATERIAL & METHOD

MATERIAL AND METHODS.

The present study was carried out in the Department of Paediatrics in collaboration with the Department of Biochemistry, M.L.B.Medical College and Hospital, Jhansi(U.P.) from May 1982 to March 1983. 48 Infants (more than 3months of age) and pre-school children (1 to 5 years of age) attending the Well Baby Clinic and those admitted in the Paediatric Ward were selected for the study.

Cases were grouped as follows :-

- A. Control group.
- B. Study group.

Selection for Control group -

10 Infants and pre-school children weighing more than 80% of the 50th percentile of Harvard Standard for their age, who were apparently healthy or convalescing after a mild attack of Polio, were selected for the Control group.

Selection for Study group -

38 Infants and pre-school children suffering

from Protein Energy Malnutrition (P E M), weighing less than 80% of the 50th percentile of Harvard Standard for their age, comprised the study group.

Children suffering from Primary liver disorders or diseases like Diabetes mellitus, Primary hypertension, Myxoedema, Renal disorders and Malaria etc. affecting the total lipids or its fractions were excluded from both the control as well as the study group.

Besides name, age, sex, address, socio-economic status, occupation of parents, educational qualifications of parents, birth order of child in the family and per-capita income, following facts were recorded in each case on specially designed proforma.

Dietary history -

A detailed dietary history since birth till the completion of this study was recorded with special emphasis on the following points.

- i) The age upto which breast milk was given.
- ii) The age when artificial milk was started, the type of artificial milk, its dilution and the average quantity consumed daily.
- iii) The age when semi solids and solids were started.

- iv) The present dietitic history in terms of quantity and quality of food used for feeding the child.

An average of total calories and protein intake per day were recorded in each case to know the quantum of deficiency for that age.

Birth history -

A detailed birth history was recorded with special emphasis on low birth weight (including prematures and small for dates).

Mile stones -

The developmental history of each child was recorded in all the four fields i.e. Gross motor, Manipulative or Fine motor, Adaptive or Social and Speech. The age at which the child attained different mile stones was ascertained by subjective and objective assessment.

Present and Past Illnesses -

From the parents or other family members, detailed history was obtained regarding present and past illnesses. Efforts were made to find out the occurrence of any acute or chronic illness like Tuberculosis, worm infestation, pertusis, measles and Malaria.

etc. that might have affected the nutritional status of the child and also the lipid and its fractions in the blood.

Family history -

An enquiry was made about the history of diabetes, tuberculosis, hypertension etc. in the family members.

All patients of study group were put on nutritional therapy to raise the daily intake of food to 200 Calories per kg. with 3-4 gms. proteins per kg. of the present body weight. (Walia and Regini, 1982), with vitamins and minerals supplementation. Drugs for infections and infestations, and intravenous fluids were used as and when required.

An attempt was made to follow up the cases upto 20 days.

Clinical examination -

A thorough clinical examination was done including general appearance, psychomotor changes, hair changes, facial look like moon facis or wizened look. Eyes were examined for the presence of conjunctival xerosis, hitot's spot, pallor and any other abnormality. Lips , gums and tongue were examined for the presence of

angular stomatitis, cheilosis, bleeding and spongy gums, glossitis etc. Total number of teeth present in the oral cavity and their health status was noted. Skin was examined for the presence of hyperkeratosis, depigmentation, xerosis and various dermatoses. Skeletal system was examined for the presence of any deformity and signs of Vitamin D deficiency such as cranic-tubes, bossing of skull, Harrison sulcus, knock knee or bow legs.

Clinical assessment for the loss of subcutaneous fat and muscle wasting was done in each child. Thyroid gland was examined to find out any abnormality.

A thorough examination was done to detect any systemic abnormality.

Anthropometric measurements -

Measurements were conducted on the day of admission, on 10th day and on 20th day.

Weight - Infant weighing scale capable of measuring weight to the nearest of 0.050 kg. was used for children who could not stand, and adult type spring weighing machine was used for recording weight nearest to 0.250 kg in elder children. Weight was recorded

with minimum clothes and before each measurement zero error was corrected. Same machines were used for subsequent followup.

Length - By using an infantometer, recumbent length was measured to the nearest of 0.1 cm., by placing the child supine on the infantometer. Head was held firmly against the fixed upright head board and legs straightened, keeping feet at right angles to legs with toe upwards. The free foot board was brought into firm contact with the child's heel.

Head circumference - With the help of a narrow flexible steel tape, head circumference was measured by applying it firmly over the glabella and supra-orbital ridges anteriorly and the occipital prominence posteriorly, giving the maximal circumference.

Chest circumference - In the mid respiration the chest circumference was measured with the help of a steel tape at the level of xiphoid cartilage in a plane at right angles to the vertebral column. The measurement was recorded in recumbent position to the nearest of 0.1 cm.

Mid arm circumference - Circumference of left upper arm at the point mid way between the tip of acromion process of scapula and olecranon process of ulna, was measured to the nearest of 0.1 cm. while the arm was hanging freely by the side.

Mid calf circumference - Maximal calf circumference was measured nearest to 0.1 cm. on the left lower limb when the child was bearing weight on it.

INVESTIGATIONS.

Each child was subjected to the following laboratory investigations.

Blood - Haemoglobin, Total and Differential leucocyte counts.

Total Serum Proteins.

Serum Albumin.

Serum Globulin.

Sugar.

Urine - Routine examination.

Stool - Routine examination for ova and Cyst.

Mantoux Test and Radiological investigations were performed when necessary.

Besides above investigations lipid status was assessed by estimating-

Total Serum Cholesterol level

Serum Total Lipids and

Serum Free Fatty Acids.

Collection of Blood samples -

Blood samples were collected from the peripheral vein or femoral vein of each child by venipuncture on the day of admission, 10th day and 20th day under all aseptic precautions and after taking consent from the parents.

Blood specimen was collected in plain vial, and was allowed to clot. The serum was then transferred to a test tube and centrifuged. The upper clear layer of serum was transferred to another test tube which was stored in deep freezer for analysis.

ESTIMATION OF TOTAL SERUM CHOLESTEROL

On the principle of Zlatkis, Zak and Boyle (1953), serum total cholesterol was estimated by Henley's method (1957).

Principle - Cholesterol in acetic acid solution gives red colour when treated with ferric chloride and sulphuric acid.

Reagents -

1. Acetic Acid - Glacial acetic acid (A.R) aldehyde free.

2. Ferric chloride reagent - 0.05% solution.

This reagent was prepared by dissolving 50 mg. of $\text{FeCl}_3 \cdot 6\text{H}_2\text{O}$ salt in 100 ml. of glacial acetic acid (A R). The prepared solution was stored at room temperature.

3. Sulphuric acid - Sulphuric acid (A R).

4. Stock cholesterol standard solution -

100 mg. of cholesterol powder was dissolved in 100 ml. of glacial acetic acid (A R) to prepare stock cholesterol standard solution. The solution thus prepared was kept in cool and dark place.

5. Cholesterol standard for use -

The above cholesterol standard solution was diluted 1 to 25 with ferric chloride reagent. This solution was also kept in cool and dark place.

Technique -

0.1 ml. of serum was added to 10 ml. of ferric chloride reagent in a test tube. The mixture was mixed well and was kept for 10-15 minutes for the proteins to flocculate. The whole mixture was centrifuged at 2,500 rpm. for 10-15 minutes and then from the clear supernatant fluid 5 ml. was transferred to another

test tube. 3 ml. of sulphuric acid (A.R) was added by the side of the test tube and the mixture was mixed thoroughly by shaking, and then allowed to stand for 20-30 minutes.

For the Standard - 0.1 ml. of physiological saline was mixed with 10 ml. of cholesterol standard solution for use and then out of this mixture 5 ml. was transferred to another test tube.

For the Blank - 5 ml. of ferric chloride reagent was taken in a third test tube.

The samples were simultaneously treated for Blank and Standard.

Values of unknown (Test Serum) and Standard were read on colorimeter against Blank to set zero using a yellow filter.

Calculations -

Total cholesterol in mg / 100 ml. serum

$$= \frac{\text{Reading of unknown}}{\text{Reading of standard}} \times \frac{100}{0.05} \times 2$$

$$= \frac{\text{Reading of unknown}}{\text{Reading of standard}} \times 400.$$

In general total cholesterol was performed on each specimen, however to test reproducibility duplicate determinations were performed at intervals throughout the study.

ESTIMATION OF SERUM TOTAL LIPIDS

Phosphovanillin method (Frings and Fendley, 1972 and Annino, 1976) was employed to estimate serum total lipids by using Span Diagnostic Kit (Art. No. 926).

Principle - Serum lipids on heating with concentrated sulphuric acid and phosphovanilline reagent produce pink colour which can be measured colorimetrically.

Reagents - The kit contained the following reagents -

1. Lipid Standard (700 mg/dl.)
2. Sulphuric acid (36 N)
3. Phosphovanilline Reagent.

Vanilline Reagent - 6.0 gms of Vanilline was dissolved in water in 1 litre volumetric flask and diluted to give a total volume of 1 litre. The solution was stored in brown bottle.

Phosphovanilline Reagent - In 350 ml of vanilline reagent 50 ml of water and 600 ml of conc. phosphoric acid was added with constant stirring in a 2 litre flask. The solution was finally stored in brown bottle in refrigerator between 2 to 8°C .

Procedure -

Estimations were carried out in test serum

alongwith blank and lipid standard.

To 2 ml of 36% sulphuric acid 0.1 ml of serum was added and mixed thoroughly. The mixture was kept in a boiling water bath for 10 min and then cooled to room temperature. From the above mixture 0.2 ml fluid was transferred to another test tube and 6.0 ml of phosphovanilline reagent was added. The mixture, thoroughly mixed was incubated at 37° C for 15 min time and then cooled to room temperature.

For standard - 0.1 ml of lipid standard and

For Blank - 0.1 ml distilled water was used.

Optical density of the test serum and standard lipid solution were measured at 540 nm by using green filter against blank to set zero.

Calculation -

$$\text{Serum total lipids in mg/dl} = \frac{\text{Reading of test}}{\text{Reading of standard}} \times 700$$

ESTIMATION OF FREE FATTY ACIDS IN SERUM

Millian Novaks technique (1965) was employed for the estimation of free fatty acids in serum.

Principle - Free fatty acids are extracted from the serum and then esterified with the help of cobalt reagent which are then estimated colorimetrically with the help of the indicator.

Reagents -

1. Cobalt reagent -

Solution A - Cobalt nitrate-acetic acid-potassium sulphate, was prepared by adding 6 gms of cobalt nitrate $\text{L CO} (\text{NO}_3)_2 \cdot 6 \text{H}_2\text{O}$ and 0.8 ml of glacial acetic acid to a saturated solution of potassium sulphate (saturated while boiling, with excessive crystals at storage, and filtered before use) to give a total volume of 100 ml at 37°C .

Solution B - A saturated sodium sulphate (Na_2SO_4) solution was prepared by adding sodium sulphate powder to boiling water. It was stored at 37°C .

Preparation of Cobalt reagent - Triethanolamine 1.35 volume was made upto 10 volumes with solution A. 7 volumes of solution B were added and then the mixture was shaken well. This reagent was prepared every time fresh for the analysis, as it was not stable.

2. Indicator -

Stock solution - 0.4 % alpha-nitroso beta-naphthol in 96 % ethanol was prepared by dissolving 0.4 gm of it in 100 ml of ethanol.

For use - 4 ml of stock indicator solution was diluted with 46 ml of 96 % ethanol.

3. Dole's extraction mixture - This mixture was prepared by mixing redistilled Isopropyl alcohol 40 parts, Heptane 10 parts and N H₂SO₄ 1 part.

4. Chloroform-Heptane solution- Redistilled chloroform and N heptane were mixed in the ratio of 5 : 1 (V/V) to prepare chloroform-heptane solution.

5. Standard Palmitic Acid solution - 0.05M palmitic acid solution was prepared by dissolving 1.3 gms of palmitic acid in 100 ml of Dole's extraction mixture and was stored at 0° C.

Procedure -

Estimations were carried out in duplicate alongwith blank and palmitic acid as the standard.

To 2.5 ml of Dole's extraction mixture in test tube 1 ml of serum was added. The liquids were mixed by shaking. The test tube was cooled for 10 min in ice cooled water. To this 3 ml heptane was added followed by 4 ml of glass distilled water. The contents were thoroughly mixed and then allowed to stand for 10 min. After the phases had separated, 2 ml was drawn from the upper heptane phase and transferred to another test tube. 4 ml of chloroform-heptane mixture was added to it followed by 5 ml of freshly prepared cobalt reagent and the solution was thoroughly mixed

for 3 min. The mixture was centrifuged for 15 min at 2,500 rpm and then 4 ml of upper chloroform-heptane phase was transferred to a test tube containing a pinch of anhydrous sodium sulphate powder. From this 3 ml of the dehydrated chloroform-heptane mixture was transferred to a test tube containing 3.5 ml of the indicator solution.

For standard- 1 ml palmitic acid solution and

For blank- 1 ml distilled water was used and these tubes were treated exactly as for serum as described above. Values were read 30 min later at 500 millimicron (m) in a spectrophotometer.

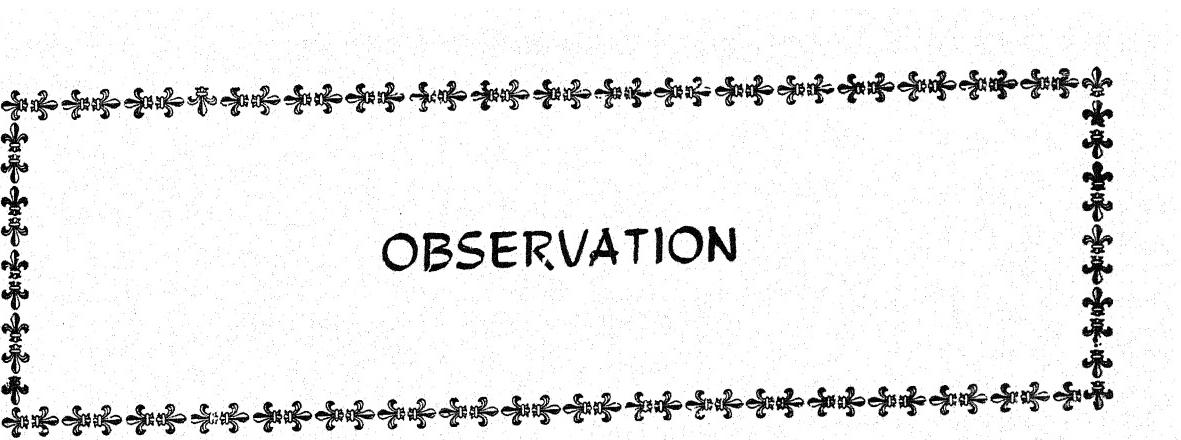
Calculations -

Standard solution : 1.3 gms of palmitic acid/dl
Dole's extraction mixture (5.07 mEq/l) .

$$\text{FFA in mEq/l} = \frac{\text{Reading of unknown (test)}}{\text{Reading of Standard solution}} \times 5.07$$

In each analysis, two standard solutions were treated simultaneously to reduce the error.

In general FFA estimation was performed on each specimen, however to test reproducibility triplicate determinations were performed at intervals throughout the study.



OBSERVATION

OBSEERVATIONS

The present study comprised of 38 malnourished and 10 healthy/convalescing poliomyelitis infants and children admitted in Paediatric ward or attending the Well Baby Clinic of M.L.B. Medical College Hospital, Jhansi (U.P.). They were classified into 4 groups according to Indian Academy of Paediatrics Classification of Malnutrition (1972). There were 2 cases of under-nutrition, 16 cases of marasmus, 15 cases of marasmic kwashiorkor and 5 cases of kwashiorkor.

The malnourished children were investigated on the day of admission, 10th day and then on 20th day of starting dietary therapy. All the children of various malnutrition groups could not be followed up to 20th day as evident from Table-2, as 3 of them expired, 2 of marasmic group and 1 of marasmic kwashiorkor group, 9 of them left against medical advice and 5 developed malaria. Undernutrition group could be followed upto 10th day only. ' Paired T test ' was used to test the statistical significance in the follow up levels ,

TABLE - 1

AGE, BODY WEIGHT AND LENGTH IN CONTROL AND STUDY GROUP CASES

CONTROLS	CONTROL GROUP	STUDY GROUP					
		UNDERNUTRITION		MARAS MUS		MARAS MTC KWASHIORKOR	
		Mean	SD	Mean	SD	Mean	SD
AGE (in months)	25.70 ± 19.21 (10)	11.90 ± 0.71 (2)		23.16 ± 13.16 (16)		23.60 ± 12.65 (15)	31.00 ± 18.46 (5)
BODY WEIGHT (in kg.)	10.55 ± 2.66 (10)	6.62 ± 0.18 (2)		5.48 ± 1.70 (16)		5.65 ± 1.39 (15)	8.70 ± 2.71 (5)
LENGTH (in cms.)	80.32 ± 11.57 (10)	69.80 ± 1.70 (2)		83.28 ± 3.41 (16)		69.26 ± 8.45 (15)	74.60 ± 10.50 (5)

TABLE - 2

SERUM TOTAL LIPIDS LEVELS IN CONTROL AND STUDY GROUP CASES

		SERUM TOTAL LIPIDS LEVELS IN mg/dl.									
		STUDY GROUP									
		CONTROL		UNDERNUTRITION		PARASITIC		KASHIYOROKO		E	
GROUPS ACCORDING TO TIME INTERVAL	Mean	A SD	B Mean	C SD	D Mean	E SD	F Mean	G SD	H Mean	I SD	J SD
1. admission	666.12 ± 47.44	491.70 ± 6.08	593.92 ± 49.52	420.45 ± 61.35	491.80 ± 41.77						
	(10)	(2)	(16)	(15)	(15)						
2. 10th DAY	-	643.23 ± 8.65	620.93 ± 43.03	674.98 ± 59.63	752.33 ± 84.47						
	-	(2)	(12)	(13)	(5)						
3. 20th DAY	-	-	604.95 ± 34.66	637.46 ± 73.68	688.40 ± 65.72						
	-	-	(9)	(9)	(4)						

groups compared :

		P values			P values			P values			P values		
		A1 vs C1	C1 vs C2	C1 vs C3	D1 vs D2	D1 vs D3	D1 vs E1	D2 vs D3	D2 vs E2	D3 vs E3	E1 vs E2	E1 vs E3	E2 vs E3
A1		0.01	0.01	0.01	0.01	0.01	0.01	0.05	0.05	0.05	0.01	0.01	0.01
A1	vs	D1	C2	C3	D2	D3	E1	D3	E2	E3	E1	E2	E3
N1		0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
N1	vs	E1	C2	C3	D2	D3	E1	D3	E2	E3	E1	E2	E3
C1		0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01
C1	vs	C2	C3	D2	D3	E1	E2	E3	E3	E3	E1	E2	E3

SERUM TOTAL LIPIDS LEVELS IN CONTROL AND STUDY GROUP

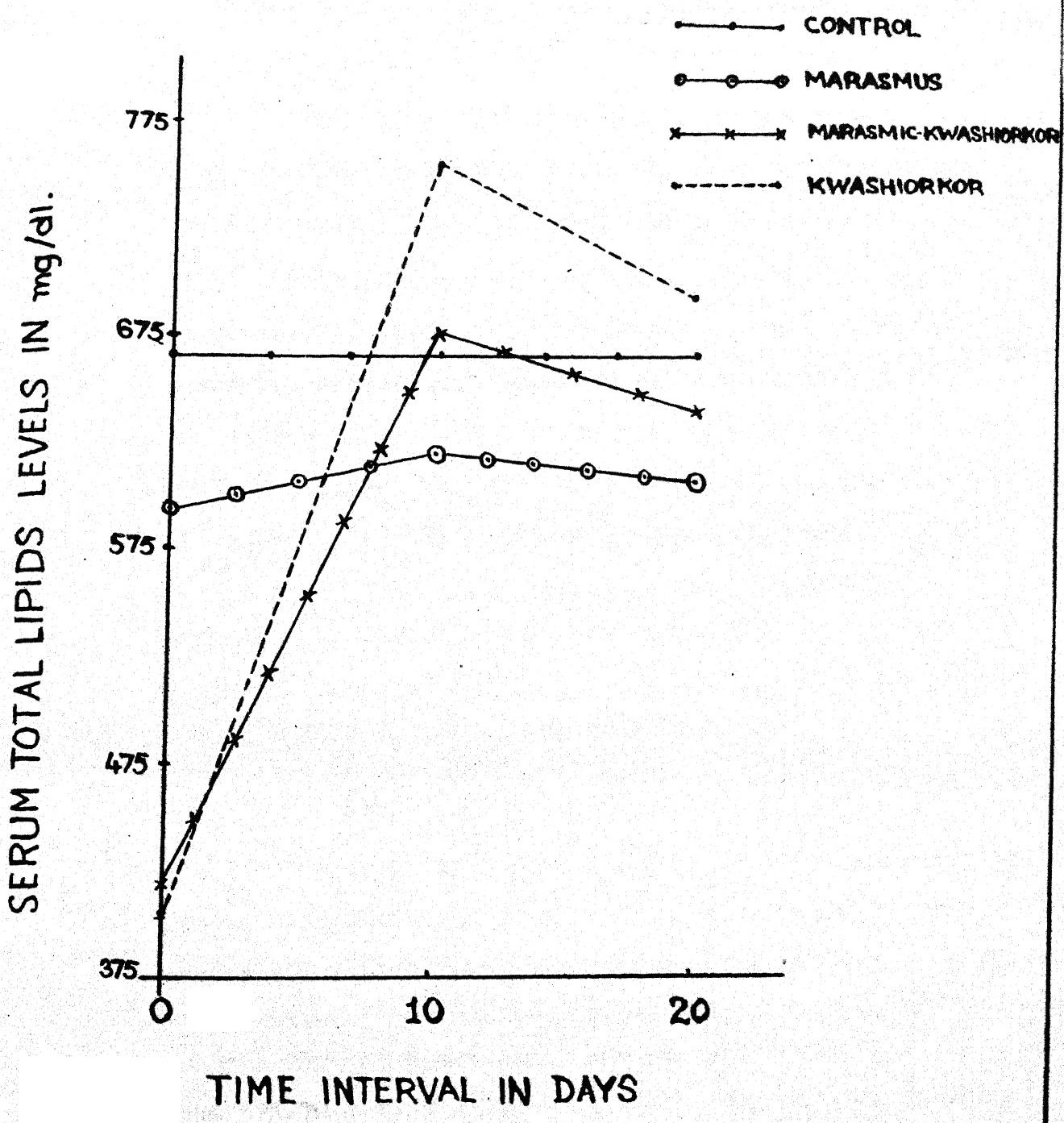


FIG. 1

' Student T test ' was used for evaluating significant difference between levels in different groups.

As evident from Table-1, the mean age in control, undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor children was 25.70 ± 19.21 , 11.50 ± 0.71 , 23.19 ± 13.16 , 23.60 ± 12.65 and 31.00 ± 18.46 months respectively. The mean body weight in undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor groups was 6.62 ± 0.18 , 5.48 ± 1.70 , 5.65 ± 1.39 and 8.70 ± 2.71 kg respectively, and mean body weight of control group was 10.55 ± 2.66 kg. The mean length in control group was 80.32 ± 11.57 cm, whereas in undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor group, the mean length was 69.90 ± 1.70 , 83.28 ± 3.41 , 69.26 ± 8.45 and 74.60 ± 10.50 cm respectively.

SERUM TOTAL LIPIDS LEVEL

Serum total lipids levels in various malnutrition groups and in control group are shown in Table-2. The mean total lipids level in control group was 666.12 ± 47.44 mg/dl, whereas in undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor groups, the levels at admission were 491.70 ± 6.08 , 593.92 ± 49.52 , 420.45 ± 61.35 and 401.50 ± 41.77 mg/dl respectively. The mean total lipid levels in control group was significantly higher than

the levels in all the malnutrition groups ($P < 0.01$).

The levels in marasmic group were significantly higher than those in marasmic kwashiorkor and kwashiorkor groups ($P < 0.01$). But the levels in later two groups were statistically not significant ($P > 0.05$).

In the marasmic group, during follow up, the mean serum total lipids levels on 10th day and on 20th day of therapy were 620.93 ± 43.03 and 604.85 ± 34.66 mg/dl respectively. The 1st and 2nd follow up levels were significantly higher as compared to the levels on admission day ($P < 0.01$), but the levels on 20th day were not significantly different from the levels on 10th day.

In the marasmic kwashiorkor group, the mean serum total lipids levels on 10th day and on 20th day of follow up were 574.98 ± 50.63 and 637.46 ± 73.68 mg/dl respectively. The serum total lipids levels at admission were significantly low as compared to the levels on 10th day and 20th day follow up ($P < 0.01$). The levels of serum total lipids on 1st follow up were significantly higher than those on 2nd follow up ($P < 0.05$).

As evident from the Table-2, in kwashiorkor group, the mean serum total lipids levels on 10th day and 20th day were 752.33 ± 84.47 and 688.40 ± 65.72 mg/dl

TABLE - 3
SERUM TOTAL CHOLESTEROL LEVELS IN CONTROL AND STUDY GROUP CASES

		SERUM TOTAL CHOLESTEROL LEVELS IN mg/dl.											
		STUDY GROUP											
		UNDEMYDURITON		MAMSMIS		MAMSMIC		KASHITOROR					
Groups	according to time interval	A	B	C	D	E	F	G	H	I	J	K	L
		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1. ADMISSION	10th day	168.25 ± 10.18	(10)	134.65 ± 16.19	(2)	145.76 ± 12.47	(16)	118.08 ± 22.69	(12)	170.60 ± 20.04	(15)	104.81 ± 8.29	(5)
2. 10th day	-	-	-	-	-	-	-	-	-	-	-	-	-
3. 20th day	-	-	-	-	-	-	-	-	-	-	-	-	-
		P values											
		P values											
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SERUM TOTAL CHOLESTEROL LEVELS IN CONTROL AND STUDY GROUP

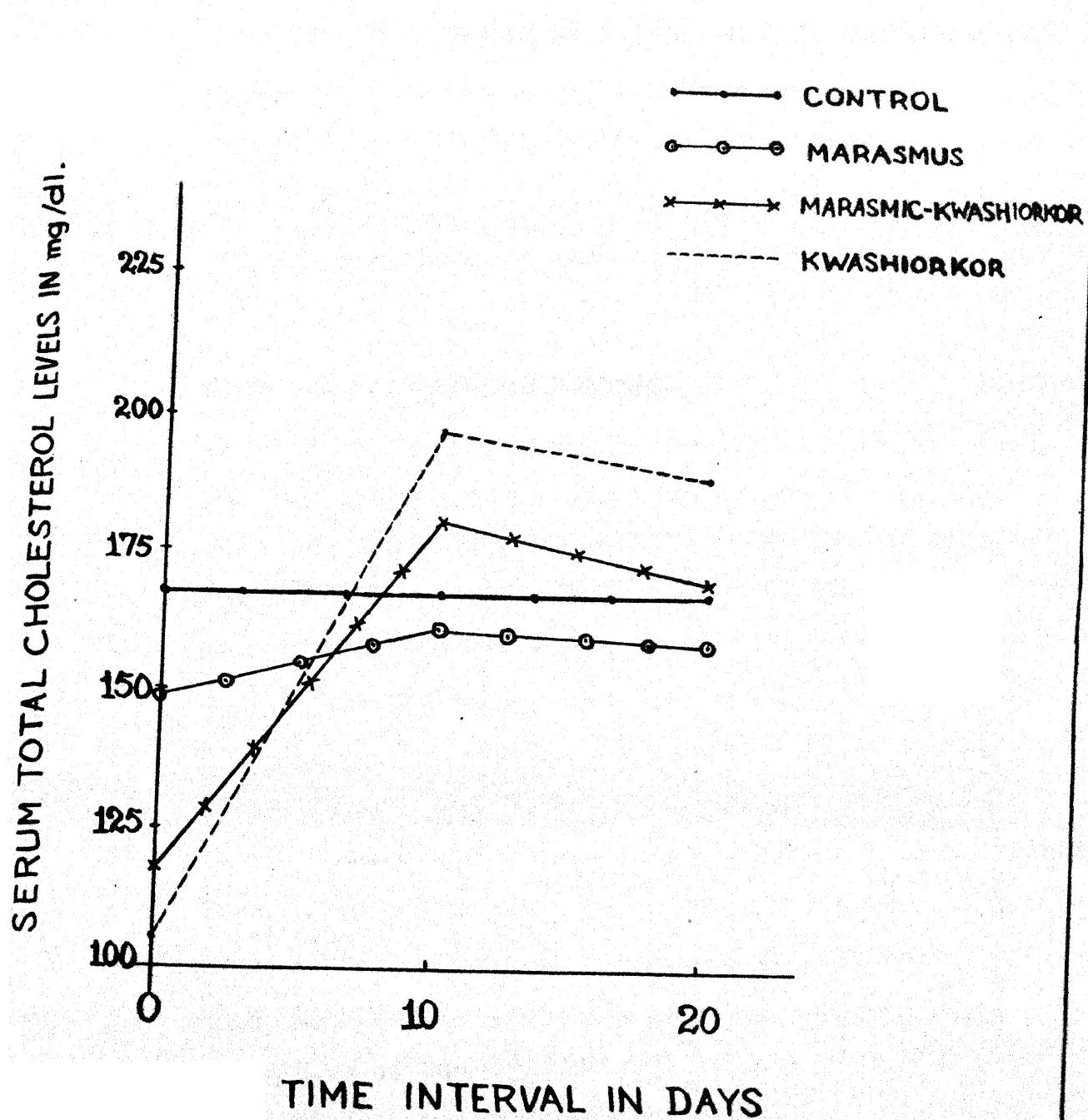


FIG. 2

respectively. The total lipids level at admission was found to be statistically significantly low as compared to the total lipids levels on 1st and 2nd follow up ($P < 0.01$). The total lipids level on 2nd follow up was not significantly different from the level on 1st follow up ($P \geq 0.05$).

SERUM TOTAL CHOLESTEROL LEVELS

Table-3 depicts serum total cholesterol levels in control and various malnourished groups. In control group the mean serum cholesterol level was 168.25 ± 10.18 mg/dl, whereas in undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor group, the mean cholesterol levels at the time of admission were 134.65 ± 16.19 , 148.76 ± 12.47 , 118.08 ± 22.69 and 104.81 ± 8.29 mg/dl respectively. The mean serum cholesterol levels in all the malnourished groups were found to be significantly lower than that in the control group ($P < 0.01$). The level in marasmic group was significantly higher than those of marasmic kwashiorkor and kwashiorkor groups ($P < 0.01$), but the levels in later two groups were statistically not different ($P \geq 0.05$).

During follow up of the marasmus group, the mean cholesterol level on 10th day and on 20th day were 162.10 ± 7.77 and 159.09 ± 9.06 mg/dl. The mean serum cholesterol levels on 1st and 2nd follow up were

TABLE - 4

SERUM FREE FATTY ACIDS LEVELS IN CONTROL AND STUDY GROUP CASES

STUDY GROUP	DETERMINATION	MATERIAL		MANUFACTURER		Mean	SD	Mean	SD	Mean	SD	P value
		C	E	D	C							
SERUM FREE FATTY ACIDS LEVELS IN nEq/l.												
1. <i>Normal</i>	1. <i>Normal</i>	0.495 ± 0.035	0.497	0.632 ± 0.037	0.632	-	-	-	-	-	-	-
2. <i>1000 DAY</i>	2. <i>1000 DAY</i>	-	-	0.465 ± 0.066	(2)	0.485 ± 0.060	(16)	0.505 ± 0.064	0.774	0.109	0.993 ± 0.097	(4)
3. <i>2000 DAY</i>	3. <i>2000 DAY</i>	-	-	0.465 ± 0.066	(2)	0.485 ± 0.060	(16)	0.520 ± 0.052	0.475	0.037	0.432 ± 0.040	(9)
4. <i>Malnutrition</i>	4. <i>Malnutrition</i>	-	-	-	-	0.520 ± 0.052	(12)	-	-	-	-	(9)
5. <i>Obese</i>	5. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
6. <i>Diabetics</i>	6. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
7. <i>Normal</i>	7. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
8. <i>Obese</i>	8. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
9. <i>Diabetics</i>	9. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
10. <i>Obstructive</i>	10. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
11. <i>Normal</i>	11. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
12. <i>Obese</i>	12. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
13. <i>Diabetics</i>	13. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
14. <i>Obstructive</i>	14. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
15. <i>Normal</i>	15. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
16. <i>Obese</i>	16. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
17. <i>Diabetics</i>	17. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
18. <i>Obstructive</i>	18. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
19. <i>Normal</i>	19. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
20. <i>Obese</i>	20. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
21. <i>Diabetics</i>	21. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
22. <i>Obstructive</i>	22. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
23. <i>Normal</i>	23. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
24. <i>Obese</i>	24. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
25. <i>Diabetics</i>	25. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
26. <i>Obstructive</i>	26. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
27. <i>Normal</i>	27. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
28. <i>Obese</i>	28. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
29. <i>Diabetics</i>	29. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
30. <i>Obstructive</i>	30. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
31. <i>Normal</i>	31. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
32. <i>Obese</i>	32. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
33. <i>Diabetics</i>	33. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
34. <i>Obstructive</i>	34. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
35. <i>Normal</i>	35. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
36. <i>Obese</i>	36. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
37. <i>Diabetics</i>	37. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
38. <i>Obstructive</i>	38. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
39. <i>Normal</i>	39. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
40. <i>Obese</i>	40. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
41. <i>Diabetics</i>	41. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
42. <i>Obstructive</i>	42. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
43. <i>Normal</i>	43. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
44. <i>Obese</i>	44. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
45. <i>Diabetics</i>	45. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
46. <i>Obstructive</i>	46. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
47. <i>Normal</i>	47. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
48. <i>Obese</i>	48. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
49. <i>Diabetics</i>	49. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
50. <i>Obstructive</i>	50. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
51. <i>Normal</i>	51. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
52. <i>Obese</i>	52. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
53. <i>Diabetics</i>	53. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
54. <i>Obstructive</i>	54. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
55. <i>Normal</i>	55. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
56. <i>Obese</i>	56. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
57. <i>Diabetics</i>	57. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
58. <i>Obstructive</i>	58. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
59. <i>Normal</i>	59. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
60. <i>Obese</i>	60. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
61. <i>Diabetics</i>	61. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
62. <i>Obstructive</i>	62. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
63. <i>Normal</i>	63. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
64. <i>Obese</i>	64. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
65. <i>Diabetics</i>	65. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
66. <i>Obstructive</i>	66. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
67. <i>Normal</i>	67. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
68. <i>Obese</i>	68. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
69. <i>Diabetics</i>	69. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
70. <i>Obstructive</i>	70. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
71. <i>Normal</i>	71. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
72. <i>Obese</i>	72. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
73. <i>Diabetics</i>	73. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
74. <i>Obstructive</i>	74. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
75. <i>Normal</i>	75. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
76. <i>Obese</i>	76. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
77. <i>Diabetics</i>	77. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
78. <i>Obstructive</i>	78. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
79. <i>Normal</i>	79. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
80. <i>Obese</i>	80. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
81. <i>Diabetics</i>	81. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
82. <i>Obstructive</i>	82. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
83. <i>Normal</i>	83. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
84. <i>Obese</i>	84. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
85. <i>Diabetics</i>	85. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
86. <i>Obstructive</i>	86. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
87. <i>Normal</i>	87. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
88. <i>Obese</i>	88. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
89. <i>Diabetics</i>	89. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
90. <i>Obstructive</i>	90. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
91. <i>Normal</i>	91. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
92. <i>Obese</i>	92. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
93. <i>Diabetics</i>	93. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
94. <i>Obstructive</i>	94. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
95. <i>Normal</i>	95. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
96. <i>Obese</i>	96. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
97. <i>Diabetics</i>	97. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
98. <i>Obstructive</i>	98. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
99. <i>Normal</i>	99. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
100. <i>Obese</i>	100. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
101. <i>Diabetics</i>	101. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
102. <i>Obstructive</i>	102. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
103. <i>Normal</i>	103. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
104. <i>Obese</i>	104. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
105. <i>Diabetics</i>	105. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
106. <i>Obstructive</i>	106. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
107. <i>Normal</i>	107. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
108. <i>Obese</i>	108. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
109. <i>Diabetics</i>	109. <i>Diabetics</i>	-	-	-	-	-	-	-	-	-	-	-
110. <i>Obstructive</i>	110. <i>Obstructive</i>	-	-	-	-	-	-	-	-	-	-	-
111. <i>Normal</i>	111. <i>Normal</i>	-	-	-	-	-	-	-	-	-	-	-
112. <i>Obese</i>	112. <i>Obese</i>	-	-	-	-	-	-	-	-	-	-	-
113. <i>Diabetics</i>	113. <i>Diabetics</i>	-	-</									

found to be significantly higher than that on admission day. There was no statistical difference observed between the levels on 10th day and on 20th day ($P \geq 0.05$).

In the marasmic kwashiorkor group, during follow up the mean total cholesterol level on 1st follow up and 2nd follow up were 181.66 ± 16.11 and 170.60 ± 20.04 mg/dl respectively. The mean serum total cholesterol level on 1st and 2nd follow up were found to be significantly higher than the admission level ($P < 0.01$). The mean total cholesterol level on 1st follow up was significantly higher than the level on 2nd follow up ($P < 0.05$).

In the kwashiorkor group, the mean cholesterol level on 10th day and on 20th day were 196.36 ± 21.22 and 189.32 ± 10.52 mg/dl. The mean total cholesterol level at admission was significantly lower than the levels on 1st follow up and 2nd follow up ($P < 0.01$). The mean cholesterol level on 1st follow up was statistically not different from the level on 2nd follow up ($P \geq 0.05$).

SERUM FREE FATTY ACIDS LEVELS

Serum free fatty acids levels in control group and various malnutrition groups are shown in Table-4. In the control group, the mean serum free

SERUM FREE FATTY ACIDS LEVELS IN CONTROL AND STUDY GROUP

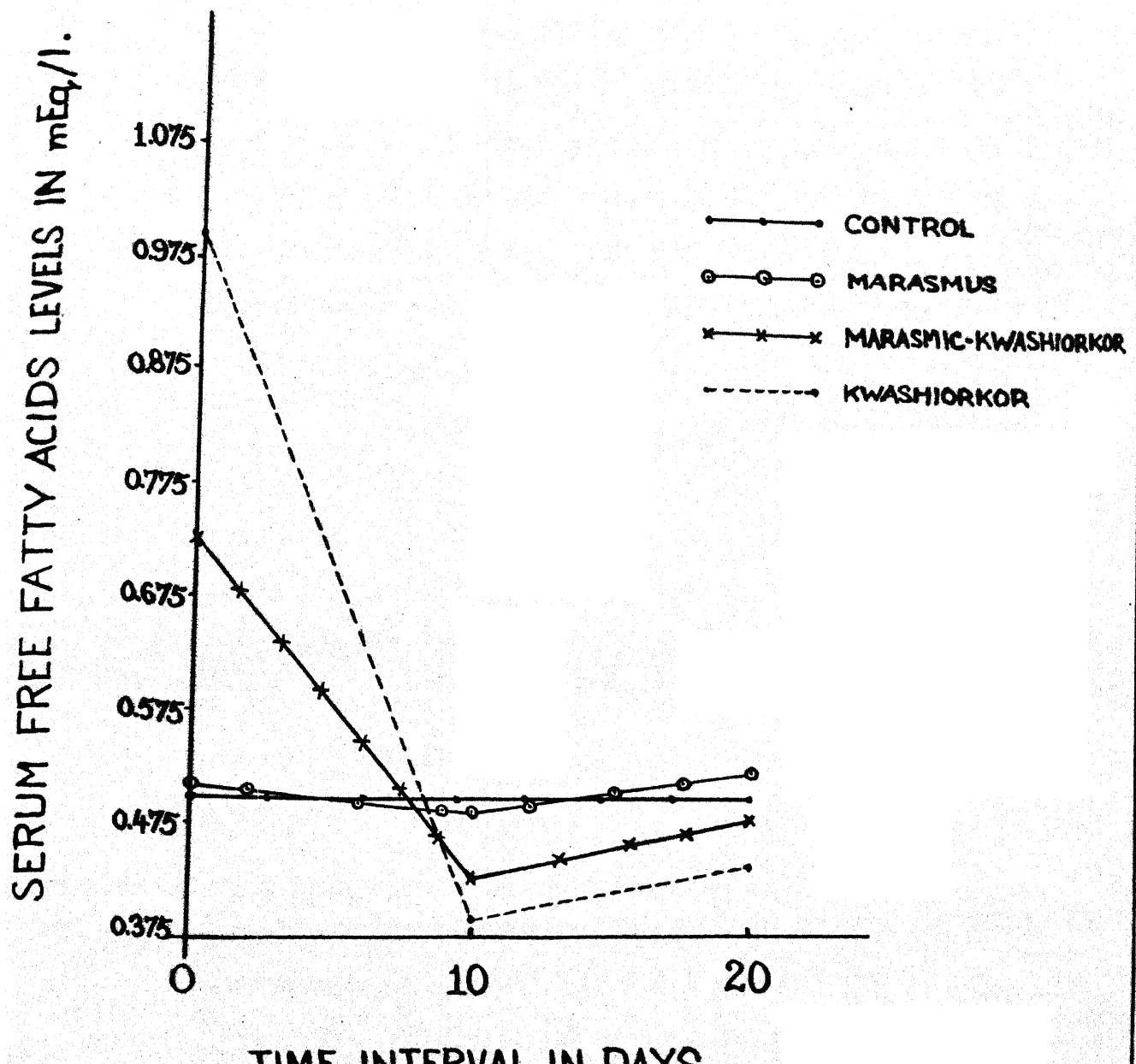


FIG. 3

fatty acids level was 0.495 ± 0.037 mEq/l , whereas at the time of admission in undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor groups, the serum free fatty acids levels were 0.632 ± 0.097 , 0.505 ± 0.064 , 0.774 ± 0.109 and 0.993 ± 0.097 mEq/l . The free fatty acids level of control group were significantly low as compared to the levels of marasmic kwashiorkor and kwashiorkor groups, but were statistically not significant when compared to the marasmus group levels ($P > 0.05$).

During follow up of marasmic cases, the mean serum free fatty acids levels on 1st follow up and 2nd follow up were 0.485 ± 0.060 and 0.520 ± 0.052 mEq/l respectively. The levels on 1st or 2nd follow up were statistically not significantly different when compared to the admission levels ($P > 0.05$). The levels on 1st and 2nd follow up were also not significantly different ($P > 0.05$).

In the marasmic kwashiorkor group, the mean serum free fatty acids levels on 10th day and on 20th day were 0.426 ± 0.047 and 0.475 ± 0.037 mEq/l respectively. The level at admission was found to be significantly higher as compared to levels on 1st and 2nd follow up ($P < 0.03$), but the difference between the later two groups was statistically insignificant ($P > 0.05$).

METHODS USED IN STUDY alone cases

In the kwashiorkor group, during follow up the mean free fatty acids levels in serum on 1st follow up and 2nd follow up were 0.389 ± 0.046 and 0.432 ± 0.040 mEq/l respectively. The levels on 1st and 2nd follow up were significantly low when compared to the initial levels ($P < 0.01$). The levels on 1st and 2nd follow up were statistically not different from each other.

WEIGHT GAIN IN STUDY GROUP

Table-5 shows the weight gain pattern in various malnutrition groups, during their follow up on 10th day and 20th day of dietary therapy.

During follow up of marasmus group, the mean weight of children on 1st and 2nd follow up were 6.01 ± 1.53 and 6.53 ± 1.13 kg respectively as compared to the weight at admission (5.48 ± 1.70 kg). A significant difference was noted between the weight at admission and weight on 1st follow up, and also between 1st follow up and 2nd follow up weight levels ($P < 0.01$).

In marasmic kwashiorkor group, the mean weight noted at 1st follow up after 10 days therapy and at 2nd follow up after 20 days therapy were 6.11 ± 1.51 and 6.70 ± 1.88 kg respectively. The difference in weight observed at admission and at 1st follow up, and between

TABLE - 6
RATE OF WEIGHT GAIN IN STUDY GROUP CASES

NO. OF CASES	MARASMUS			MARASMIC KWASHIORKOR			KWASHIORKOR		
	Mean	SD	N	Mean	SD	N	Mean	SD	N
MEAN GAIN IN WEIGHT (gm / kg / day)									
16	14.41	2.89		9.66	5.08	15	5.57	2.47	5
GROUPS COMPARED :									
MARASMUS	vs	MARASMIC KWASHIORKOR		<	0.05				
MARASMUS	vs	KWASHIORKOR		<	0.01				
MARASMIC KWASHIORKOR	vs	KWASHIORKOR	7	0.05					

1st follow up and 2nd follow up was statistically significant ($P < 0.01$).

In the kwashiorkor group during follow up, the mean weight observed on 1st and 2nd follow up were 9.04 ± 2.70 and 8.42 ± 1.23 kg respectively. The difference observed between the weight at admission and on 1st follow up, weight at admission and weight on 2nd follow up, and weights on 1st and 2nd follow up were statistically significant ($P < 0.01$).

RATE OF WEIGHT GAIN

Table-6 shows the rate of weight gain per unit of body weight per day in marasmus, marasmic kwashiorkor and kwashiorkor groups from the time of admission to the completion of study. The rate of weight gain in marasmus group was found to be 14.41 ± 2.81 gm/kg/day, whereas in the marasmic kwashiorkor and kwashiorkor groups, the rates of weight gain were 9.66 ± 5.08 and 5.57 ± 2.47 gm/kg/day respectively. A significant difference was noted in the rates of weight gain in marasmic group and marasmic kwashiorkor group ($P < 0.05$), and a highly significant difference was observed between the rates of weight gain in marasmus group and kwashiorkor group ($P < 0.01$). No significant difference was noted in the rates of weight gain in marasmic kwashiorkor and kwashiorkor groups ($P > 0.05$).

TABLE - 7SERUM ALBUMIN LEVELS IN CONTROL AND STUDY GROUP CASES

GROUPS ACCORDING TO TIME INTERVAL	SERUM ALBUMIN LEVELS IN gm / dl.									
	CONTROL		STUDY GROUP							
	GROUP	UNDERNUTRITION	MARASMC	MARASMC	KWASHIORKOR	KWASHIORKOR	KWASHIORKOR	KWASHIORKOR	KWASHIORKOR	KWASHIORKOR
A	B	C	D	E	F	G	H	I	J	K
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	SD
1. ADMISSION	4.06 ± 0.42 (10)	3.95 ± 0.35 (2)	3.29 ± 0.44 (16)	2.23 ± 0.42 (15)	2.16 ± 0.39 (5)					
2. 10th DAY	-	3.30 ± 0.35 (2)	3.81 ± 0.14 (12)	3.27 ± 0.40 (13)	3.16 ± 0.30 (5)					
3. 20th DAY	-	-	4.07 ± 0.14 (6)	3.95 ± 0.31 (9)	3.90 ± 0.42 (4)					

Groups compared:		P value	P value
A1	Vs C1	≤ 0.01	≤ 0.01
A1	Vs D1	≤ 0.01	≤ 0.01
A1	Vs E1	≤ 0.01	≤ 0.01
C1	Vs C2	≤ 0.01	≤ 0.01
A1	Vs E2	≤ 0.05	≤ 0.05
A1	Vs E3	≤ 0.05	≤ 0.05

SERUM ALBUMIN LEVELS IN CONTROL AND STUDY GROUP

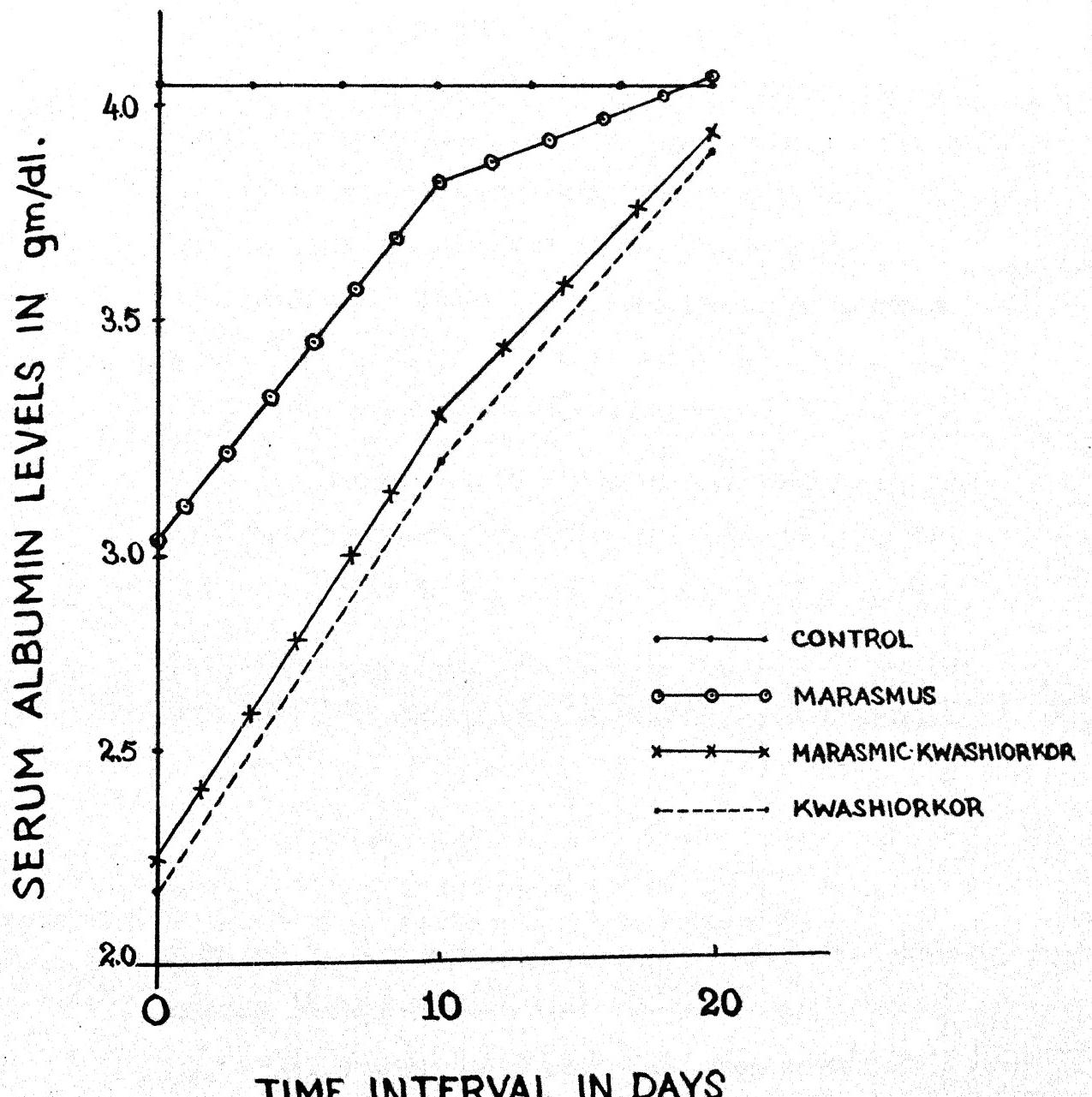


FIG. 4

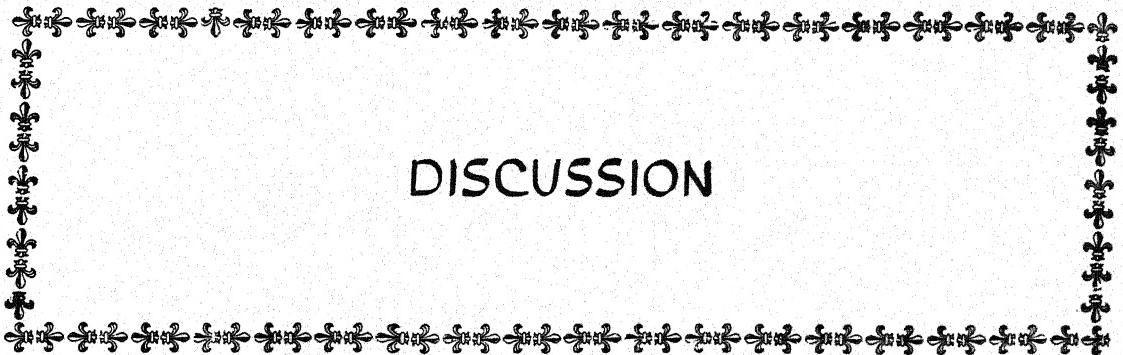
SERUM ALBUMIN LEVELS

Table-7 shows the serum albumin levels in control and various study groups. In the control group , the mean serum albumin level was 4.06 ± 0.42 gm/dl, whereas the levels of serum albumin at the admission time in undernutrition, marasmus, marasmic kwashiorkor and kwashiorkor groups were 3.25 ± 0.35 , 3.29 ± 0.44 , 2.23 ± 0.42 and 2.16 ± 0.30 gm/dl respectively. There was a significant difference observed between the levels in control groups and those in all the malnutrition groups ($P < 0.01$).

During the follow up of marasmic group, the mean serum albumin levels at 1st follow up and 2nd follow up were 3.81 ± 0.14 and 4.07 ± 0.14 gm/dl respectively. A significant difference was observed between the level at admission and 1st follow up and also between 1st follow up and 2nd follow up ($P < 0.01$).

In the marasmic kwashiorkor group, the serum albumin levels on 1st follow up and 2nd follow up were 3.27 ± 0.40 and 3.95 ± 0.31 gm/dl respectively. The serum albumin levels at admission and at 1st and 2nd follow up were significantly different from each other ($P < 0.01$).

In the kwashiorkor group, the mean serum albumin levels on 1st and 2nd follow up were 3.16 ± 0.30 and 3.90 ± 0.43 gm/dl respectively and the albumin level at admission time was 2.16 ± 0.30 gm/dl. The difference between the initial and 1st follow up level was highly significant ($P < 0.01$) and between 1st and 2nd follow up was significant ($P < 0.05$).



DISCUSSION

DISCUSSION

The present study was carried out on 38 malnourished and 10 control infants and children. Children suffering from primary liver disorders or diseases like Diabetes mellitus, Primary hypertension, Myxoedema, Renal disorders and Malaria etc. affecting the total lipids or its fractions, were not included in the control as well as the study group. Observations have been presented in Table 1 to 7. A critical analysis of our data with tangible inference is dealt herewith.

In the undernutrition group, which comprises the biggest group of malnourished population, only 2 cases were admitted for the study, as it was difficult to convince the parents for the admission and the sick children of this group admitted were suffering from diseases affecting the lipid status, so an appreciable number could not be included in the present study. These cases, though investigated, were not included for statistical analysis.

The mean age of marasmus group in our study was 23.1° months, similar to the observation made by Lewis et al (1964). Gurson and Saner (1969) Rao et al (1969), Agbedana (1979) have reported the mean age in marasmus to be between 1-2 years, but Mc Laren (1966) and Hijazi (1974) reported the mean age as less than 1 year. Mc Laren (1966) suggested that early and abrupt weaning associated with diluted and unhygienic formulae, repeated infections and starvation therapy leads to nutritional marasmus in infancy. In our study besides other social factors, prolonged breast feeding (upto 16 months , range 4-36 months) and delayed weaning could be the important causes of the higher mean age of marasmus group.

In our study, the mean age of the kwashiorkor group was 31 months, which was similar to the finding of Agbedana (1979). The range of age noted by Ramaathan (1955), Schwartz and Dean (1957), MacDonald et al (1963), Lewis et al (1964 , 1966), Jaya Rao and Krishna Prasad (1966), Mc Laren (1966), Rao et al (1969), Agbedana (1979) was 1-3 years. The probable explanation of higher mean age being that kwashiorkor is a nutritional imbalance due to low

protein and rich carbohydrate diet frequently as a result of prolonged breast feeding and weaning on to traditional starchy family diet, prevalent in this area.

During follow up of the marasmic group after starting the dietary therapy, the mean weight rose significantly from the pretreatment level to the level on 1st follow up and continued to rise significantly till 2nd follow up (Table-5). Brooke and Wheeler (1976) recorded a more pronounced rise in weights, but their follow up weights were recorded on 2nd week and 4th week after dietary therapy.

In the kwashiorkor group, after starting the dietary therapy, the weight rose significantly from the pretreatment stage to 2nd follow up in each case. One of the 5 cases of kwashiorkor left against medical advice after 1st follow up, therefore the mean weight of remaining 4 cases, as shown in Table-5, appears to be apparently lower than the initial mean weight of 5 cases. Brooke and Wheeler (1976) in their study, noted an initial fall in the weight on 1st follow up, which was due to loss of oedema, then the weight started rising, as noted on 2nd follow up after 4 weeks of dietary therapy. In our study,

it took on an average 7 days time for oedema to disappear and then the kwashiorkor cases started gaining weight.

In the present study, in the marasmic kwashiorkor group, the weight started rising significantly from the time of admission to the 1st follow up and continued till 2nd follow up. The pattern of weight gain in our study was similar to the pattern noted by Brooke and Wheeler (1976) and Kumar et al (1983).

In marasmic group, the rate of weight gain in our study was significantly higher than those in kwashiorkor and marasmic kwashiorkor groups (Table-6). Waterlow (1961) and Ashworth (1962) have also reported similar finding in their studies on weight gain. In the kwashiorkor group, the rate of weight gain in our study was not significantly different from that in the marasmic kwashiorkor group. Suraj Gupte (1979) reported that after initial 10-14 days of dietary therapy, the rate of weight gain in both the marasmic and kwashiorkor group is same i.e. 10-15 gm/day. Lily Philip et al (1982) observed a difference in weight gain in

marasmic children if Nicotinic acid was added to the diet. In the children without Nicotinic acid in the diet, rate of weight gain was 171.81 gm/kg/month, whereas in those children, in whom Nicotinic acid was added, in the diet, rate of weight gain was 231.05 gm/kg/month.

In the present study serum total lipid levels of the control group were higher than those of all the malnourished groups (Table-2 Fig.1). Schwartz and Dean (1957), Cravioto et al (1959) and MacDonald et al (1963) reported low levels of serum total lipids in kwashiorkor cases at the time of admission, whereas Van Der Sar (1951) reported variable levels in kwashiorkor. Contrary to our findings Hansen (1958), Monckberg (1968) and Gurson et al (1973) reported normal levels of total lipids in marasmus groups at the time of admission.

In kwashiorkor, prolonged poor nutrition results in reduction in the intestinal absorption of different fat fractions (Viteri, Flores and Behar, 1966) which is further reduced by attacks of diarrhea. These children also have an abnormally high content of free bile acids throughout the small intestine and show distorted intestinal villi with a change in

structure from finger like to leaf and convoluted villi (Schneider and Viteri, 1972). Both malnutrition and diarrhea also produce a decrease in concentration of conjugated bile acids, which are essential for solubilization of lipids in the intestinal lumen and their absorption through formation of lipid micelles (Schneider and Viteri, 1974 a). The transport of fat from the intestine to the liver is not primarily altered (Viteri and Schneider, 1974) but the transport of fat from the liver to the tissues as low density lipoproteins is markedly reduced, because of impaired production of the protein moiety of β lipoproteins.

The fatty liver, characteristic of kwashiorkor is perhaps due to increased fat transport from the adipose tissues to liver (Lewis et al, 1964) to decreased β lipoproteins synthesis (Flores et al, 1970) and possibly due to increased liver lipogenesis (Metcoff, 1975).

Schwartz and Dean (1957), Cravioto et al. (1959) and MacDonald (1963) suggested that the low levels of total lipids were due to reduced levels of triglycerides, cholesterol, phospholipids and

lipoproteins. During the active stage of disease in kwashiorkor group, Sriniwasan and Patvardhan (1952) observed decreased activity of plasma esterase, lipase and amylases, which results in low levels of total lipids.

In our study, the serum total lipid levels at the time of admission in marasmic group were found to be significantly higher than in the kwashiorkor group (Table-2). This observation was consistent with the finding reported by Schwartz and Dean (1957), Cravioto et al (1959), MacDonald et al (1963), Hansen (1968), Monckberg (1968) and Gurson et al (1973). Gurson et al (1973) suggested that similar to mechanisms controlling proteins and carbohydrate homeostasis, the marasmic infants probably maintain a balanced source for lipids as a result of which the plasma levels of total lipids remain within normal limits.

Serum total lipid levels in the marasmic kwashiorkor group were found to be , as expected, in between the marasmic and kwashiorkor levels. In our study these levels were significantly different from the control and marasmic levels, but were insignificant when compared to the kwashiorkor group (Table-2 Fig-1). This marasmic kwashiorkor group

forms a major group of the malnourished population in this region. Serum total lipid levels in this group have not been described separately by other workers. Malnutrition cases with oedema have been either described as Kwashiorkor (Williams, 1933 ; Davies, 1948 ; Van Der Sar, 1951; Dean and Schwartz , 1953 ; Schwartz and Dean 1957 , etc.) or Nutritional oedema syndrome (Gopalan and Patwardhan, 1951 , Venkatachalam, Sri Kantia and Gopalan, 1954).

In the marasmus group, the serum total lipid levels showed a significant rise on 1st follow up and then there was an insignificant fall on 2nd follow up as compared to the 1st. Hansen (1968) Monckberg (1968) and Gurson et al (1973) have reported no significant change in the follow up levels in marasmus group. It is to be recalled that pretreatment total lipid values were also not different from the controls. Gurson et al (1973) have reported low levels of phospholipids at admission as compared to controls, in follow up they found further decrease in the phospholipids. Reduction of this fraction of total lipids may be cause of reduced total lipid levels in our study.

In the present study in the kwashiorkor , the serum total lipid level at the time of admission was significantly low as compared to controls,which rose significantly on the 10th day of therapy and then fell insignificantly on 20th day , but the level still remained significantly higher than the level at admission time. Our observation was consistent with the observations made by Schwartz and Dean (1957), Cravioto et al (1959) and MacDonald et al (1963). Schwartz and Dean (1957) explained the rise in total lipid levels after dietary therapy to be due to sudden increase in neutral fat and esterified cholesterol levels which was to some extent brought by the diet provided, but mainly it was due to the release of preformed fat and cholesterol from the stores. The fall in the level of total lipids that succeeded the initial rise presumably represented utilisation of fat in a normal manner. Cravioto et al (1959) compared the changes occurring in the lipids during recovery from kwashiorkor to those of the new born babies, and found a striking resemblance. They further observed that these changes being independent of the amount and the kind of the diet consumed and also of the age and sex of the patients. MacDonald et al (1963) suggested that the increase in the total lipid levels was due to the increase in

cholesterol, glycerides and phospholipids, and the fall in the total lipid levels after 10 days therapy was almost entirely due to the sharp fall in glyceride fraction.

In the marasmic kwashiorkor group, in the present study, the total lipids rose significantly from the initial level to reach to the control level on 10th day and then showed a fall though insignificant . The observed values were in between the marasmus and kwashiorkor group values.

In the present study serum cholesterol levels in all the malnourished groups were lower than the control group. Our finding was consistent with the observations made by Van Der Sar (1951), Dean and Schwartz (1953) , Workers from Nutrition Research lab. Coonoor South India (1954) Rammathan (1955), Schwartz and Dean (1957), Cravioto et al (1959), Schendel and Hansen (1961), MacDonald et al (1963), Lewis et al (1964) Jaya Rao and Krishna Prasad (1966), Taylor (1971), Debnath (1972) and Agbedana (1979) in kwashiorkor cases. Our findings were similar to the findings noted by Truswell and Hansen (1969) and Debnath (1972) in marasmus cases. The various explanations suggested for the decreased cholesterol levels in kwashiorkor are decreased activity of plasma

esterase, lipase and pancreatic amylase in the children in active stage of kwashiorkor (Srinivasan and Patvardhan 1952), accumulation of fat and cholesterol in liver and other stores in the tissues (Dean and Schwartz, 1953 Nutrition Research Workers, Coonoor, 1954, Ramnathan, 1955, Schwartz and Dean 1957, Schendal and Hansen, 1961), defective transport of fat from liver to tissues due to lack of appropriate protein acceptors (Debnath, 1972).

Truswell and Hansen attributed decreased levels of plasma lipoproteins to be the cause of low level of cholesterol in marasmus. Debnath (1972) suggested that poor nutrition results in reduced amount of body fat, reduced endogenous fat metabolism leading to hypocholesterolaemia. Agbedana (1979) suggested a defective mobilization of liver lipids to be a cause of decreased cholesterol levels in plasma and thought that decreased hepatic lipoprotein lipase may be responsible for it. Lewis et al (1964) and Gurson et al (1973) found normal levels of serum cholesterol in the marasmic group. Gurson et al (1973) suggested that marasmic cases probably maintain a balanced source for lipids as a result of which the plasma levels of cholesterol remain within normal limit.

The serum cholesterol levels at admission in marasmic group were found to be significantly higher than in the kwashiorkor and marasmic kwashiorkor groups. In our study, our finding was similar to the findings reported by Van Der Sar (1951), Dean and Schwartz (1953), Workers from Nutrition Research lab. Coonoor (1954), Rammathan (1955) etc.

The serum cholesterol level in the kwashiorkor group and marasmic kwashiorkor group were insignificantly different. The serum cholesterol level in the marasmic group was significantly lower than the level in control group (Table-3, Fig.2), similar observation was made by Debnath (1972). After 10 days of dietary therapy the levels increased and came to the control levels and then persisted at this level even on 20th day of therapy. The pattern of changes in cholesterol levels in marasmic group, observed in our study, was consistent with the pattern observed by Debnath (1972), Lewis et al (1964) and Gurson et al (1973) on the other hand, noted no significant changes in the serum cholesterol levels before or after therapy.

In the kwashiorkor group , the serum cholesterol level, before starting the dietary therapy was significantly low as compared to the level in control group. Our finding was similar to the findings

noted by all the previous workers. After initiating the dietary therapy, the cholesterol levels increased significantly on 10th day and crossed the control levels and remained so till our 2nd follow up.

Similar observations have been noted by Dean and Schwartz (1953), Nutrition Research lab. Cooneer (1954), Schwartz and Dean (1957), Schendel and Hansen (1961), MacDonald et al (1963), Lewis et al (1964), Jaya Rao and Krishna Prasad (1966), Debnath (1972) and Agbedana (1979).

Schendel and Hansen (1961) reported that the rise in cholesterol level appeared to be maximal during the first two weeks of adequate dietary treatment and they suggested that the serum concentration of cholesterol reflect the rate at which liver fat is cleared from stores during recovery. They further observed that an arrested rise or fall was associated with the onset of complications. or inadequate therapy. Contrary to the explanation given by Schwartz and Dean (1957), Jaya Rao and Krishna Prasad (1966) attributed the rise in cholesterol level solely to the caloric intake. Debnath (1972) suggested that the protein in the diet helped in the discharging lipids from the liver in the form of lipoproteins.

Schwartz and Dean (1957) MacDonald et al (1963), Lewis et al (1964), Jaya Rao and Krishna

Prasad (1966) and Debnath (1972) reported a return of serum cholesterol levels to the control level on 20th day of therapy, whereas similar to our findings , Rammathan (1955) Schendel and Hansen (1961), Taylor (1971), noted persistently higher levels even on 20th day of therapy, thus showing a delayed fall in the cholesterol levels.

In the marasmic kwashiorkor group the serum cholesterol level was significantly lower than the control group level in the pretreatment phase and it was not significantly different from the level in kwashiorkor group. On 1st follow up the cholesterol level rose significantly to a level higher than the control level and then on 2nd follow up the level fell and came to the control group level.

In the present study , serum free fatty acid levels in the kwashiorkor and marasmic kwashiorkor groups were found to be significantly higher than in the control group. The free fatty acid levels in the marasmic group were insignificantly different from the control levels. Higher levels of serum free fatty acids in kwashiorkor group before starting the therapy, have been noted by Lewis et al (1964, 1966) Jaya Rao and Krishna Prasad (1966) Hadden (1967) Agbedana (1979) . Hadden (1967), Gurson and Saner (1969)

Gurson et al (1973) and Agbedana (1979) reported normal levels of serum free fatty acids in marasmic group. Lewis et al (1964, 66) reported higher levels of free fatty acids in serum in marasmic children and suggested that the rate of release of free fatty acids from adipose tissue is reciprocally related to the extent of carbohydrate utilization. Glucose administration depresses the free fatty acid production and plasma free fatty acid levels, whereas prolonged starvation is associated with elevated plasma levels.

They further suggested that in kwashiorkor calorie deficiency is responsible for the rise in free fatty acid levels. They divided the course of kwashiorkor into two phases-a chronic phase characterized by low protein, relatively adequate carbohydrate diet, advancing protein depletion and reasonable caloric balance and a later acute phase in which reduced intake and malabsorption produced a state of general under nutrition. In acute stage there is little fat synthesis from carbohydrate which is otherwise seen at adipose organs in early phase. Lewis et al (1964) concluded that increased flux of fatty acids from adipose tissue to the liver was responsible for the elevated plasma free fatty acid levels. Jaya Rao and Krishna Prasad suggested that the high circulating levels of non-esterified fatty acids could be due to hepatic damage

causing defective uptake of nonesterified fatty acids or that nonesterified fatty acid levels represented an alternate source of energy to tissues.

Hadden (1967) suggested a block at the point of entry of short chain of fatty acyl coenzyme A into the Kreb cycle and temporary impairment of carbohydrate tolerance as responsible for the high levels of free fatty acids. Agbedana (1979) proposed the cause for high levels of free fatty acids in serum to be due to effect of growth hormone and insulin in kwashiorkor.

Serum free fatty acid levels in marasmic group were found to be significantly lower than in the kwashiorkor and marasmic kwashiorkor group. Our observation was similar to the findings reported by Hadden (1967) and Agbedana (1979). It could be because the children of kwashiorkor and marasmic kwashiorkor group face more stressful situation than marasmus and probably because of the same reason , the free fatty acid levels in kwashiorkor group were significantly higher than in the marasmic kwashiorkor group.

In the present study the serum free fatty acid level at the time of admission in marasmic group was not significantly different from the control level

(Table-4, Fig. 3). After starting the dietary therapy , on 10th day and on 20th day the levels remain unchanged. A similar pattern was observed by Hadden (1967), Gursen and Saner (1969), Gursen et al (1973). As in kwashiorkor, high levels of free fatty acids were reported in marasmus before starting dietary therapy, by Lewis et al (1964), which came down drastically on 1st follow up and then rose to the control level on 2nd follow up.

In the kwashiorkor group, the free fatty acid levels in serum were significantly higher than the control group which after instituting the dietary therapy, fell significantly to a level lower than the control on 1st follow up and then rose insignificantly on further follow up. Similar observation was noted by Lewis et al (1964), Jaya Rao and Krishna Prasad (1966) and Hadden (1967). Lewis et al (1964) Proposed that the calorie mainly in the form of carbohydrate provided in the kwashiorkor cases was responsible for the sudden fall in free fatty acid levels. After 20 days therapy the levels were insignificantly different from the control levels. Jaya Rao and Krishna Prasad (1966) suggested that after starting the therapy the defective uptake of free fatty acids by the liver might have corrected.

thus resulting in sudden fall in nonesterified fatty acid levels.

In the marasmic kwashiorkor group before starting the dietary therapy, in our study the free fatty acid levels were significantly higher than the control group levels and were in between the marasmic and kwashiorkor group levels. After 10 days of dietary therapy the levels fell significantly and then showed an in-significant rise to come to the control levels. The pattern was similar to the pattern observed in kwashiorkor cases.

In our study, serum albumin levels in the control group were found to be significantly higher than those in all the malnourished groups (Table-7, Fig.4). Our finding was consistent with the findings of Lewis et al (1966) and Debnath (1972). In kwashiorkor group, Ramaathan (1955) and Macdonald et al (1963) have reported low serum albumin levels in the pretreatment phase. Gurson et al (1973) have noted low levels of serum albumin in marasmic groups.

Hypoalbuminaemia has been attributed as a basic change in kwashiorkor. Cohan and Hansen (1962) suggested that the total albumin pool is decreased, the intravascular compartment being less affected than the extravascular. The changes in albumin concent-

ration were supposed to be the consequence of decreased rate of synthesis, which occurs before the rate of catabolism becomes reduced. James and Hay (1968) reported that in kwashiorkor the albumin turnover is lower than normal and the half life is prolonged. The other important causes of low albumin levels in kwashiorkor are - the protein turnover rate in muscles is low, synthesis rate is decreased while that of catabolism is low when carbohydrate intake is enough to suppress gluconeogenesis and normal if energy deficiency is there (Felig et al 1969 , Waterlow, 1975) and the low levels of plasma amino acids.

In our study the serum albumin levels in pretreatment stage of marasmic group, was found to be significantly higher than the levels in kwashiorkor and marasmic kwashiorkor group (Fig.4). Our finding was similar to the finding noted by Lewis et al (1964) and Debnath (1972). Serum albumin levels though higher in marasmic kwashiorkor group, were not statistically different than the levels in kwashiorkor group.

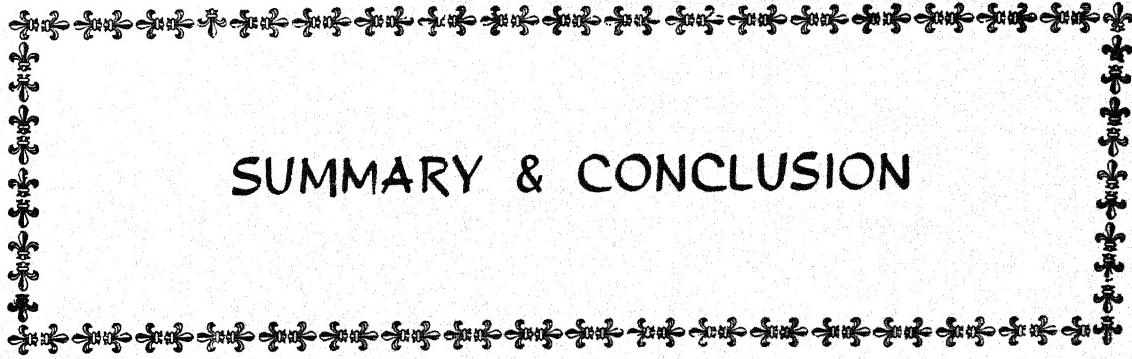
In the present study the serum albumin levels in marasmic group, after starting the therapy, rose significantly to reach the control levels on 1st follow up and then continued to rise. When paired

T test was applied the difference between the levels on 1st and 2nd follow up were significantly different. Lewis et al (1964), Debnath (1972) and Gurson (1973) also noted a progressive rise in the serum albumin levels in marasmic group. Lewis et al (1964) and Debnath (1972) did not compare their values of serum albumin with the control levels, and Gurson et al (1973) studied the albumin levels before starting the therapy and after completing the therapy (21 to 37 days).

In our study in the kwashiorkor group, the mean serum albumin level observed before starting the therapy were comparable with the findings of Ranganathan (1955), McDonald et al (1963) and Debnath (1972). These serum albumin levels in our study rose significantly on 1st follow up and continued to rise till reached to a level, insignificantly different from the control level on 2nd follow up. The rising pattern of serum albumin in our study was similar to the patterns observed by Ranganathan (1955), McDonald et al (1963), Lewis et al (1964) and Debnath (1972). The level achieved on 2nd follow up in our study was similar to the level observed by McDonald et al (1963), but was higher than those observed by Ranganathan (1955).

Lewis et al (1964) and Debnath (1972).

In the marasmic kwashiorkor group in our study, the serum albumin levels rose progressively and significantly from the pretreatment level to the control level in 20 days after starting the dietary therapy. The rising pattern in this group was similar to the pattern in kwashiorkor group.



SUMMARY & CONCLUSION

SUMMARY AND CONCLUSION

38 malnourished infants and children with varying degrees of malnutrition and 10 control children were studied for the lipid profile (Serum total lipids, Serum total cholesterol and Serum free fatty acid levels) at the severe stage and during recovery phase after starting the dietary therapy. Children were classified into 4 groups according to Indian Academy of Paediatrics Classification of Malnutrition (1972). Children suffering from Primary liver disorders or diseases like Diabetes mellitus, Primary hypertension, Myxoedema, Renal disorders and Malaria etc., affecting the total lipids or its fractions were not included in the control as well as the study group. Serum Total Lipids , Total Cholesterol and Free fatty acid levels were investigated in the malnourished children, on the day of admission, 10th day and then on 20th day of starting dietary therapy.

Serum Total lipid levels were estimated by Phosphovanilline method (Span Diagnostic Kit, Art. No. 926), Serum Total cholesterol levels by Henley's method (1957) and Serum Free fatty acid levels by Millian Novak's technique (1965).

In the present study, the mean age of marasmus group,marasmic kwashiorkor group and kwashiorkor group was 23.19 , 23.60 and 31.00 months respectively. The mean weights in the above groups were 5.48 , 5.65 and 8.70 kg respectively. All malnourished children showed a significant rise in their weights throughout the period of study and their rate of weight gain was 14.41 , 9.66 and 5.57 gm/kg/day in marasmus, kwashiorkor and marasmic kwashiorkor respectively.

Serum total lipid levels in all the malnourished groups (593.92 , 420.45 and 401.90 mg/dl in marasmic, marasmic kwashiorkor and kwashiorkor groups respectively) were significantly lower than the levels in the control group i.e. 666.12 mg/dl. During follow up , in the marasmus group, the total lipid levels showed a significant rise on 1st follow up,as levels came to 620.93 mg/dl , and then

there was an insignificant fall on 2nd follow up.

In kwashiorkor group, total lipid levels rose significantly on 10th day of therapy and came to control level on 20th day. In the marasmic kwashiorkor group the levels rose significantly and came to the control level on 10th day.

In the present study, serum total cholesterol levels in marasmus, marasmic kwashiorkor and kwashiorkor groups were 148.76 , 118.08 and 104.81 mg/dl respectively, which were significantly lower than that in the control group value (168.25 mg/dl). The total cholesterol level in the marasmus group was significantly higher than in the kwashior and marasmic kwashior groups. After starting the dietary therapy, serum cholesterol levels in the marasmic group came to the control level on 10th day. In the kwashiorkor group, the level rose significantly on 10th day and crossed the control levels and remained so till 2nd follow up , whereas in the marasmic kwashiorkor group , the levels came to the control level on 2nd follow up.

Serum free fatty acid levels in the kwashiorkor and marasmic kwashiorkor groups were 0.993 and 0.774 mEq/l respectively , which were significantly higher than the control and marasmic group levels of 0.495 and

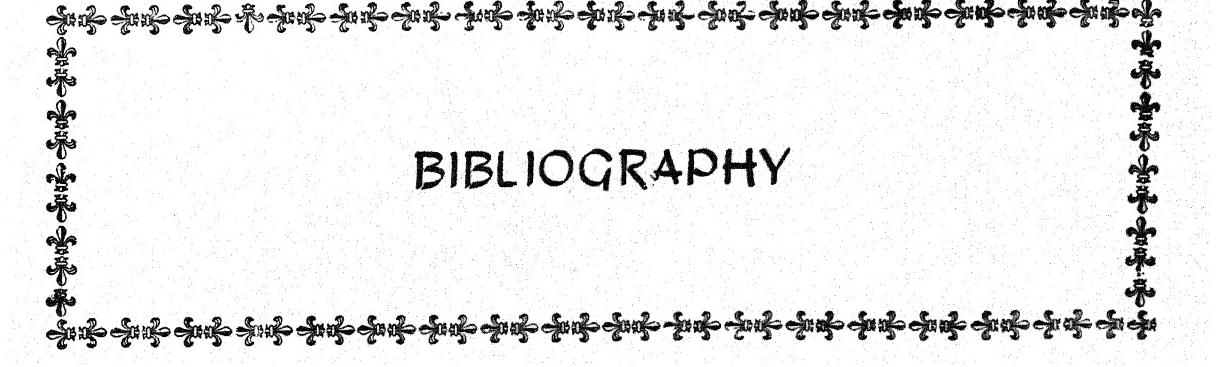
0.505 mEq/l respectively . In the marasmic group, after starting the dietary therapy no significant changes in the free fatty acid levels were found. Kwashiorkor and marasmic kwashiorkor children showed a significant fall in the free fatty acid levels on 10th and on 20th day.

Serum albumin levels in all the malnourished groups (3.29 , 2.23 and 2.16 gm/dl in marasmus , marasmic kwashiorkor and kwashiorkor groups respectively). were significantly lower than those in the control group (4.06 gm/dl). serum albumin levels in pre-treatment stage ⁱⁿ marasmic group were found to be significantly higher than the levels in kwashiorkor and marasmic kwashiorker groups. Serum albumin levels in marasmic children rose significantly to reach the control level on 1st follow up, after starting the dietary therapy. By 20th day the serum albumin levels came to the control levels in kwashiorker and marasmic kwashiorkor children.

TO CONCLUDE -

- In this Bundelkhand region, marasmic kwashiorkor forms a major malnourished group.
- Weight of the child alone is not a good index of assessing the severity of malnutrition.

- Anthropometry, clinical presentation and Biochemical changes form the ideal combination for assessing the severity of malnutrition.
- Kwashiorkor is the severest form of malnutrition , as lipid profile is maximally disturbed.
- After proper dietary treatment, though the abnormal lipid profile of malnourished children improve by 20th day, these children still remain grossly underweight and retarded in growth and development.
- Marasmus is the compensated form of malnutrition , where lipid profile is minimally disturbed.
- Marasmic kwashiorkor is a group showing a mixed pattern of changes in the lipid profile, but pattern is more close to the kwashiorkor group at the severe stage as well as during the recovery stage.
- Regression of biochemical changes to normal is the first indication of recovery from malnutrition state.



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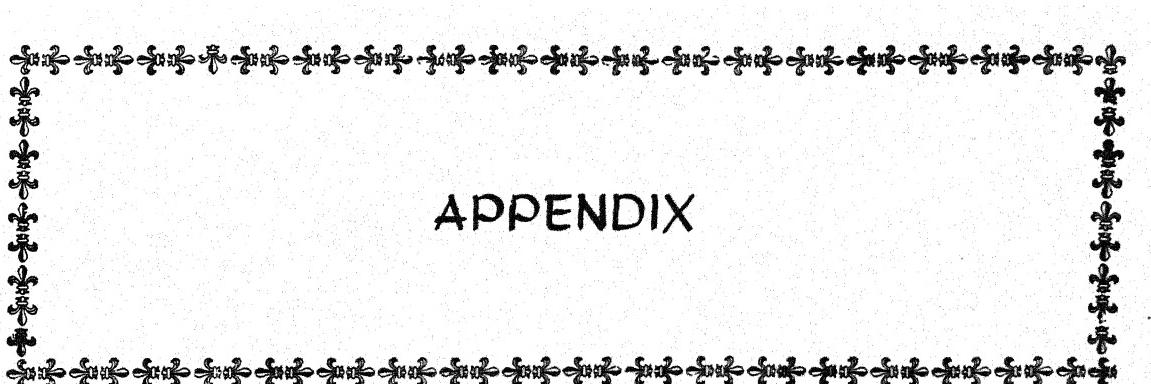
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APPENDIX

APPENDIXCASE - SHEETSl.No.**" LIPID PROFILE IN MALNUTRITION "**

Name :

Sex: M/F

Approximate date of birth:

Father's name :

Address :

Occupation:

Father

Mother

Educational qualification:

	Nil	Pri. Sch.	Matrix	Pre Univ	Grad- uate	Post grad.
Father						
Mother						

Total family income : Rs

/month

No. of family members
Above 12 years
below 12 years

Per capita income :

Geniological tree :

Dietary history :

	Started at age	Up to age	Dilution	Type
breast milk				
breast milk + artificial milk				
Artificial milk only				
Solids				

Present diet :

Calories

Proteins

Adequate/
inadequate

Immunization history :

	Age		Age
Small pox		B.C.G.	
Polio I		D.P.T I	
II		II	
III		III	
Booster I		Booster I	
IV		IV	

Antenatal history :

Gestation period	Significant illnesses (Exanthematous fever, A.P.H.Trauma Eclampsia, hypertension, Tuberculosis, Diabetes, Heart diseases etc.)	Drug intake/ radiation

Natal history:

Mode of delivery

N/ C / P / B/T

N/o birth asoxia

Post natal history (First 4 weeks):

No problem Fever Sepais Jaundice Cyanosis Others

mile stones (developmental behaviour) :

Motor:

Age

Head control

sitting

crawling

**Standing with support
without support**

Walking

Running

Comments if any -

Manipulative:

Grasp

Self feeding - Cup

Spoon

Help in dressing

Comments if any

Social :

Smile (Social)

Response to call by name

Sphincter control :	Bladder	Day	Night
	Bowel	Day	Night

Comments if any-

Speech :

Single word

Small broken sentences

Jargon speech

Long sentences .

Comments if any -

Family history :

Present illness

Past illness :

History of - Malaria (in recent past/Primary complex/
pertusis/measles/Worm infestation/Jaundice/
others.

	Clinical Examination Date			
	1	2	3	4
<u>General appearance :</u>				
Healthy
Malnourished
<u>Psychomotor changes:</u>				
Fretful
Listless
<u>Hair :</u>				
Normal
Dyspigmentation
Easy pluckability
Sparseness
<u>Face :</u>				
Moon face
Wizened look
<u>Eyes :</u>				
Conjunctival xerosis
Bitot's spot
Pale conjunctiva
<u>Mouth:</u>				
Angular stomatitis
Cheilosis
Glossitis
Swollen bleeding gums.
<u>Dentition :</u>				

Thyroid gland :**Goitre**Skin :**Oedema****Follicular hyperkeratosis****Pellagrous dermatosis****Flaky-paint dermatosis****Diffuse depigmentation****Mosaic dermatosis**Subcutaneous fat loss :**Grade I****II****III****IV**Muscle wasting :Skeleton :**Epiphyseal enlargement
(wrist)****Rickety rosary****Ant. fontanel - open/closed****Harrison's sulcus****Bossing of skull****Knock knees****Bow legs****Note -**Abdomen :**Pot belly****Liver****Spleen****Any abnormality**G.V.S. :**Normal****Any abnormality**

C.N.S :

Normal

Any abnormality

Anthropometry :

Weight

Length/height

Head circumference

Chest circumference

Mid arm circumference

Mid calf circumference

Note -

Investigations

Date

Bloods

Haemoglobin

T.L.C.

D.L.C.

Liver function
tests

Blood sugar R

UrineStool :Hb test :X-ray chest:

Serum cholesterol (Total)						
Serum total lipids						
Serum free fatty acids						
Any other investigation						

